Clinical Examination of Critically Ill Patients

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Natesh Prabu R
Bhuvana Krishna

Forewords
Bala Venkatesh
JV Peter

JAYPEE

Clinical Examination of
Critically Ill Patients

DEPARTMENT OF CRITICAL CARE MEDICINE
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HE SHALL LIVE BECAUSE OF ME
जीविष्यति मयैवासौ

Clinical Examination of
Critically Ill Patients

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Dedicated to

My family, whose unwavering support sustained me
My teachers, who ignited my passion for knowledge
My department and institution for a supportive environment and fostering excellence.

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Foreword

The history and clinical examination form an essential part of the assessment of every patient that allows the clinician to formulate a differential diagnosis, order appropriate investigations to arrive at a final diagnosis, and institute definitive treatment.

Physical examination is taught in medical schools but is often focused on an ambulatory, stable patient in whom a detailed history is available. In the intensive care unit (ICU) environment, clinical examination can be more challenging as the patient has a high illness acuity and is often surrounded by a myriad of monitors, and equipment such as ventilators, dialysis, and extracorporeal membrane oxygenation (ECMO) machines. Access to the patient can also be limited by dressings, drains, and lines. Whilst the broad principles of physical examination techniques are relevant and apply in the ICU setting, a standard physical examination as traditionally taught is not always possible in critically ill patients. Nevertheless, for example, information from hemodynamic monitoring such as central venous pressure and the waveforms may add to the assessment of the jugular venous pressure (JVP); data from a pulmonary artery catheter can aid the clinical assessment of the severity of cardiac and valvular dysfunction. The role of clinical examination in critically ill patients has been less well studied, although evidence exists to support its usefulness.

Few books have been devoted exclusively to the physical examination of a critically ill patient. The book *Clinical Examination of Critically Ill Patients* is a tremendous addition to the literature, and Drs Natesh Prabu R and Bhuvana Krishna should be congratulated on having undertaken this major work. This multiauthor book has 42 chapters and is divided into 4 sections—General Approach to Critically Ill Patients, Systemic Examination, Short and Focused Examination, and Problem-based Application and Case-based with Examples. Every chapter is written by an experienced clinician, and a large number of authors serve as examiners for the critical care training programs. Each chapter in the book begins with a brief case history followed by a systematic approach to the examination of the relevant system. The book covers the breadth of clinical practice in intensive care—the acutely unwell ICU patient, the deteriorating patient on the ward, the obstetric patient, and the organ donor—to list a few examples. The chapters on assessing acutely unwell patients during telehealth and those suffering the ill effects of prolonged ICU stay and chronic critical illness and follow-up in the clinic are unique.

At a time when several educators worldwide highlight concerns about the diminishing emphasis on physical examination and the shift toward test-centered medicine, this book is timely. There are concerns that the increasing reliance on the patient chart, or the laboratory investigations, creates what has been dubbed an “iPatient”. Clinical examination not only enables the formulation of the diagnostic approach and minimizes unnecessary investigations but is also a key to strengthening the patient–doctor relationship. This book with its patient-centered approach will be a valuable resource for trainees, educators, and clinicians practicing in intensive care.

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Foreword

It is a privilege to be asked to write a foreword to the book *Clinical Examination of Critically Ill Patient*. This book marks an important addition to critical care resources in a country that has birthed this specialty several decades after other specialties developed in India.

The approach to diagnosis in clinical medicine has followed traditional lines, which includes a detailed history, thorough examination, and then a list of differentials followed by the ordering of relevant tests, which confirm or rule out a diagnosis. This approach is not suited to a critically ill patient because of the nature of the illness (acuteness) and the need for timeliness of response (due to severely deranged physiology). Thus, in a critically ill patient, history, examination, investigations, diagnosis, and treatment often happen in parallel. The clinical examination also needs to be focused and prioritized to aspects that require urgent attention, with repeated assessments until the entire diagnosis and treatment plans are established.

This book thus fills the gap in this important facet of critical care medicine. It not only provides chapters on a systematic and clear approach to history and clinical examination in a critically ill patient but also has sections on short and focused examination of patients with disease states such as circulatory shock, poisoning, burns or trauma, as well as the problem-based approach to clinical situations that are unique in the intensive care unit (ICU) such as weaning difficulty, refractory hypoxemia, or oliguria.

This textbook will not only help novices in critical care but also the experienced clinicians to refine their approach to the evaluation of a critically ill patient since it covers a range of topics that are seen in day-to-day practice in the ICU.

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Preface

Dear Readers,

The book is unique in itself as it focuses on the systematic way of examining critically ill patients. We have many standard books for clinical examination of medical and surgical patients admitted to the hospital or visiting the outpatient departments where the things mostly become targeted. But examining a critically ill patient not only demands swift wholesome examination of the patient but also calls for simultaneous management as time is very crucial.

Our book not only covers the long-forgotten art of case-based history taking, which gives us crucial pieces of information but also a systematic way of examining all the systems of patients so that not a stone is left unturned. Each topic is backed by a case scenario that connects the readers to their real-life experiences which further helps them understand things better. The chapters are written by experienced teachers, who have given their pearls of wisdom to trainees for a systematic and complete examination.

This book has four sections. *Section 1* focuses on a general approach to all critically ill patients. It gives an overview of how the patients should be examined systematically, providing an outline that can be adapted to all patients. *Section 2* focuses on the systemic examination of critically ill patients about the concerned organ system. It provides an overview of how a single organ system is examined systematically. This includes the examination of the patient, surroundings, monitors, and utilization of ultrasonography, imaging services, etc. *Section 3* focuses on a rapid and focused approach to all critically ill patients. It gives an overview of how the patient's clinical examination is focused and rapid during emergencies or crises. *Section 4* focuses on the problem-oriented approach. This represents the application of the previous three sections, where learned principles/concepts are applied to specific situations. This division of sections helps the reader grasp the basic principles of clinical examination, from focused examination to its application.

The book will be useful for trainees of various specialties who take care of critically ill patients. During the exit examinations of Fellowships, DM Critical Care Medicine, and DrNB Critical Care, the clinical examination of patients forms a key aspect of the assessment. We hope that this book may serve as a guide for all trainees, and the book will be a resource for postgraduate students and practicing critical care physicians.

Natesh Prabu R
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We are delighted to express our heartfelt gratitude to everyone who played a significant role in making this book. We extend our deepest appreciation to all the authors of this textbook whose expertise, dedication, and commitment to disseminating knowledge have made this unique book possible. We thank M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India for collaborating with us, sharing their publishing expertise, and believing in this project. Their professionalism and guidance have been very crucial. We are extremely grateful to our family for their patience, and constant support during the process.

We are grateful to all our teachers for inspiring us not only to learn but also to share our knowledge with others. We thank our colleagues, the department and the hospital for their unwavering support. Last but not least, we would like to appreciate and thank all our patients who have served as a continual source of learning and taught us all these years. The clinical insights and real-world experiences born while treating them have made our textbook more relevant and impactful.

Once again, we thank you all for being part of this endeavor.

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Chapter 1

History Taking of a Critically Ill Patient

Sriram Sampath

Elicitation of history and formal presentation: are they important in current critical care praxis?

ABSTRACT

Rapid diagnostic techniques, and therapeutic interventions at the bedside in an intensive care unit (ICU), have wrongly decreased the value of the art and skill of history elicitation and presentation. Skillful history taking often uncovers underlying causal mechanisms. Deeper understanding needs alternative and counterfactual hypotheses which are provided by applying critical thinking as part of history taking.

INTRODUCTION

The Traditional Style of Presentation of History

The history may be presented in the following manner, "67-year-old male who lives with his wife and two children and consumes a mixed diet...has had a fever for 3 days not associated with chills, fever is intermittent, vomiting nonprojectile, which consisted of food particles." Such a start to a presentation would probably delight an Oslerian physician, but most critical care physicians would find it verbose and lacking in necessary detail.

The Algorithmic Reductionism Style

The history is more often presented in this manner, "67-year-old who lives with a partner, has had a fever for 3 days and presented to emergency. Sepsis was suspected and severe sepsis guidelines were followed and due to respiratory failure, intubation and mechanical ventilation were carried out. Acute respiratory distress syndrome (ARDS) guidelines were followed due to hypoxia, but multiorgan dysfunction and refractory shock could not be reversed. The Indian Society of Critical Care Medicine (ISCCM) End-of-Life Care (EOLC) guidelines were followed and the patient is on a comfort care pathway."

The above presentations are representative and are examples to show what should be avoided in the elicitation of history and presentation. However, most presentations may have elements of one or both styles. Practitioners of critical care would probably find both styles unacceptable but would find it difficult to formally define an acceptable style. What are the reasons that we find these styles unsatisfactory?

What should be the Style of History Elicitation and Presentation?

The traditional style was well suited to disease processes, which evolved gradually in time and complexity. Emphasis was placed on detailed descriptions of symptoms that had evolved over time, and the aim was to capture and portray pathognomonic symptoms. Etiological and pathological differential diagnoses were constructed from history and clinical examination. Diagnostic and therapeutic interventions were not emphasized, nor were they part of the process. This was probably appropriate in times when investigations and interventions were limited.

A significant proportion of current intensive care unit (ICU) admissions involve subjects in whom organ dysfunction has rapidly set in a backdrop of multiple comorbidities. These comorbidities, either singly, or in varying combinations, provide multiple competing etiological hypotheses. The undergraduate and postgraduate teaching and exit examinations do not cover these types of disease presentations sufficiently. Traditional medical teaching has emphasized "Occam's razor"—the single simple explanation is the most likely diagnosis. Most ICU patients have had many diagnostic and therapeutic interventions prior to ICU admission. Knowledge of these interventions must be incorporated into the history to aid in accurate diagnoses. Hickam's dictum, "The patient can have as many diseases (diagnoses) as she pleases," may be more appropriate for ICU patients. The traditional framework of history-taking is not suitable for the current praxis without significant modifications. Critical care trainees will have to unlearn or modify the foundational frameworks embedded in undergraduate and postgraduate training.

The algorithmic style attempts to overcome the shortcomings of the traditional style of history elicitation. It captures temporal sequences of diagnostic and therapeutic interventions. Worsening of the patients' condition is presented as triumphant technological interventions of ever-increasing complexity. Every step of these processes is governed by algorithms of impassive objectivity, which are accurately presented in history. This style or elements of this style are often used by trainees who have internalized the traditional system and replaced pathognomonic symptoms with algorithmic protocols. This style can completely ignore causation and causal mechanisms. The traditional system with all its shortcomings never ignored causation. The traditional style of history elicitation encouraged the generation of causal hypotheses as interventions were limited. An etiological diagnosis was needed in addition to a pathological diagnosis. In the current algorithmic style, the indication for interventions is mistakenly thought to be causative. Differential diagnoses of "multi-organ dysfunction with refractory septic shock" are deemed acceptable. What constitutes an acceptable history elicitation suitable for critical care has not been well studied and described.

BARRIERS TO ACCURATE AND USEFUL ELICITATION OF HISTORY

The practice of medicine is culture specific. The elicitation of history is culture and language specific. The intensivist eliciting the history and the informant supplying it are both products of their sociocultural background. Eliciting a detailed and nuanced history requires sufficient familiarity with the informant's background. Wrongly adapting the informant's phrases to standard critical care terminology can lead to delay and wasteful interventions as the case below will show:

"A 60-year-old male with a fever of 3 days, burning micturition, and hypotension was evaluated…Clinical examination did not reveal any septic focus, a computed tomography (CT) scan of the abdomen did not show any obstructive uropathy. Broad-spectrum antibiotics have been given for probable urosepsis and these will be rationalized after the arrival of culture reports." This terse and confident presentation declares that clinical and diagnostic investigations have been done for suspected urinary infection (implicitly defined). It also reassures that therapy will be more focused after culture results. This presentation has two very major omissions: (1) The diagnosis of urinary infection is based on what symptoms and how probable is the diagnosis. (2) What are the alternative hypotheses and how probable are they? The seasoned intensivist at the bedside after listening to this intervention-rich presentation is likely to be skeptical of the diagnosis. The presentation does not correspond to her "gestalt" of urosepsis in the ICU. She retakes the history from the informant and finds that when the patient had a fever he reported that his "urine was warm." Lack of cultural awareness had transcribed this to "burning micturition," and urosepsis and a CT scan abdomen were the consequences. If the CT scan had shown gallstones, a causation-free diagnosis of "cholelithiasis, biliary sepsis, and urosepsis" would have been inevitable. An accurate and useful history needs more than translatory linguistic skills, it needs a sociocultural understanding and has to generate causative and alternative hypotheses supported by symptoms accurately elicited from informants.

The method of processing historical information can also lead to errors in diagnosis and therapy. Few intensivists are objective, rational, and capable of "critical thinking" at all times. The following real-life example will show how history elicitation can be flawed: *"a 38-year-old with fever for 3 weeks presented in altered sensorium and hypotension for 1 day. He had been seen in his village 3 weeks ago and was diagnosed as dengue. A week later in a nursing home, blood tests were done, and the platelet count was low—he was diagnosed with dengue and thrombocytopenia. As he did not get better, he was admitted in the district hospital where dengue with bicytopenia (anemia and low platelets) was diagnosed. As he worsened, he was shifted to our ICU. The CT brain for altered sensorium was normal, he was on* *inotropes and broad-spectrum antibiotics for septic shock complicating dengue with bicytopenia. Cerebrospinal fluid (CSF) examination has been deferred due to the low platelet count. Even though cephalosporins are associated with thrombocytopenia the risk–benefit analysis favors the use of such antibiotics."* This presentation and the working diagnoses would be thought unsatisfactory by most intensivists.

Why is this presentation and plan unsatisfactory? The presenter seems to have fluently presented reasons for the omission and commission of diagnostic and therapeutic interventions. This is a good example of poor decision-making due to "confirmation bias." Confirmation bias is a systematic failure in thinking in which new additional information is interpreted to support a pre-existing (false) belief. In this case, every test and complication was attributed to a prior belief—"dengue," which was probably false. Confirmation bias can be difficult to overcome; in this case, on hearing the presentation by the trainee, the consultant asked, "have you seen dengue with fever for 3 weeks?," which resulted in a reply—"dengue complicated by secondary infection." A further query "Why is the patient anemic in dengue?" was answered by "hematoma due to low platelets." Dislodging the effects of confirmation bias can be difficult. In this case, physical examination revealed adenopathy and hepatosplenomegaly, which was also attributed to dengue. Only the presence of myeloblasts in the peripheral smear allowed the diagnosis of dengue to be reluctantly discarded. This case also highlights the fact that information and diagnoses can be transferred from caregiver to caregiver without validity being checked.

How could a useful and accurate history be elicited? In modern critical care, leisurely presentations and discussions are no longer possible. However, complex the case, a succinct history has to be presented within 5–10 minutes. The history taking and presentation have to serve the purposes of the patient and the duties of the critical care physician. What are the concerns and duties of an intensivist in a large teaching hospital serving patients with limited resources? Her responsibilities fall under three categories:

1. *Patient background/interaction/prognostication:* She needs to extract from the history the social and economic background of the patient and family and their expectations. She should be aware of the activities of daily life prior to illness and who are the decision-makers in the family.
2. *Diagnosis and management:* She has to make sure that the diagnosis is reasonable and appropriate management has been initiated.
3. *Trainee education:* She has to make sure that the student learns "critical thinking" and does not fall into the common error of equating skillful imitation of practices with learning.

HISTORY OF THE BACKGROUND OF PATIENT/FAMILY AND SOCIOECONOMIC ENVIRONMENT

The history should always be elicited from family members who have intimate knowledge of events. Quite often a relative who is familiar with the hospital/fluent in the language comes forward and gives information that can be misleading. A real-life example is given as follows:

"A 56-year-old male with no comorbidities presented with vomiting and breathing difficulty for 1 day. He was seen in the emergency with respiratory failure, intubated, and shifted to ICU. Chest X-ray shows pneumonia, and antibiotics for severe community-acquired pneumonia were initiated. He had altered sensorium probably septic encephalopathy/sedation/drugs from intubation." Readers will recognize elements of "algorithmic presentation" and "confirmation bias" and lack of a clear causal mechanism. The consultant who was listening had the following queries: (1) Why should a healthy person vomit and get respiratory failure in 1 day? (2) What was the cause of the vomiting? and (3) Why is he still in altered sensorium? History was retaken from the wife of the patient who stated, "I found him lying down unresponsive in a pool of vomit, later in the ambulance he woke up and complained of severe neck pain...". An urgent CT scan showed subarachnoid hemorrhage and the X-ray was reinterpreted as aspiration pneumonia. Delay and error could have been avoided by insisting that details of history be recounted from the wife rather than some distant relative who was more fluent.

The activities of daily living (ADL) should be accurately obtained. A patient who can walk 500 m will have a better prognosis in ICU when compared with a patient who is breathless on going to the toilet from the bedroom. It is essential that ADL be objectively assessed. Relatives will confidently assert that "father is very strong and fit" and in the next sentence state that he is now housebound due to dyspnea on minimal exertion. These pieces of information when captured will be of great prognostic significance when interacting with relatives.

What is the source of income, what is the economic reserve, and who are the decision-makers are important points to be elicited. Initial explorations of family dynamics are also essential as the consultant will need this information when counseling families on a day-to-day basis. Familiarity with the sociocultural environment is essential for the elicitation of useful social history. The consultant when going for the initial family discussion should be aware of all these ramifications.

HISTORY THAT IS RELEVANT TO DIAGNOSIS AND MANAGEMENT AND TRAINEE EDUCATION

History taking relevant to diagnosis in critical care has not been comprehensively dealt with in standard textbooks of critical care. A prescriptive treatment of history taking technique is not feasible as the topic is vast and the techniques are not generalizable due to sociocultural differences. A precondition to learning history-taking skills should be recognition of the trainee's strengths and weaknesses. The current critical care trainee is well-versed in rote memorization. The trainee has skills in answering multiple choice questions—selecting a correct answer from given answers to a framed question. Meaningful history-taking involves the framing of appropriate questions and the analysis of unfamiliar answers. "Critical thinking" must be learned and practiced for history taking and management. There are many definitions of critical thinking, and most definitions will include these elements:

- Information externally gathered by observation, communication, or internally from reasoning and experience
- Formally synthesize, analyze, conceptualize, interpret, or infer from this information
- Develop from above a hypothesis or a plan for action

Glaser has defined critical thinking as "A persistent effort to examine any belief or supposed form of knowledge in the light of the evidence that supports or refutes it and the further conclusions to which it tends."

Table 1 shows the application of critical thinking principles to history-taking in the three cases discussed earlier.

With increasing experience in history-taking, differential diagnoses can be created within minutes of

TABLE 1: Principles of critical thinking to history taking.

	"Urosepsis" case	***"Dengue" case***	***"Pneumonia" case***
Recognize problem	Fever hypotension of unknown cause and not urosepsis	Fever of 3 weeks? etiology	Vomiting ± altered sensorium? etiology
Priority in history	Identify symptoms that will point to etiology	Identify symptoms that will point to etiology	Identify symptoms that will point to etiology
What was relevant information?	Lack of relevant information to explain problem	Fever and bicytopenia	Vomiting unexplained with altered sensorium
Recognize unstated assumptions	Urosepsis will explain all	Dengue will explain the symptoms	Symptoms compatible with pneumonia
Understand and use language clearly	"Burning micturition" wrong translation	Dengue faithfully transcribed hospital to hospital	Pneumonia caused by vomiting—erroneous
Data interpretation	CT normal, culture awaited	Data forced to confirm dengue	Cause of pneumonia unclear
Logical relation between events	No logical chain	No logical chain	No logical chain
Logical conclusion	Septic shock without proof	Complicated dengue	Complicated pneumonia
Test conclusion	No proof	No proof	No proof
Does it make sense?	Rethink needed	Rethink needed	Rethink needed
Is it accurate?	No	No	No
What was the alternative?	Undetermined	Hematological malignancy	Subarachnoid bleed
Is the alternative explanation preferable?	Yes	Yes	Yes

(CT: computed tomography)

listening to the history. Increasing practice and skill in history taking leads to "pictures" formed in memory, which can be used in instantaneous pattern recognitions to confirm or refute etiological hypotheses. Critical thinking is however still essential to verify assumptions and manage uncommon presentations. What are the stages of evolution from a novice to an expert history taker?

- *From unconscious incompetence to conscious competence:* This would generally correspond to the training period in critical care. The trainee listens and internalizes and classifies many symptom presentations into pathognomonic patterns for future use. At this stage in training, slavish adoption of either an over-simplistic ("Occam's razor") or needlessly complicated (Hickam's) style should be discouraged. A large mental library of patterns must be systematically accumulated for future use.
- *Development of unconscious competence:* This would correspond to the post-training/early career stage of an intensivist. Nearly all cases are familiar and easily recognized as common patterns of history. Skill development continues with recognizing uncommon presentations of common diseases and rare diseases. This is also a stage where overconfidence in diagnostic ability can lead to unacceptable results.
- *Development of expertise:* This stage in its fully developed form has two characteristics. The first would be the ability to see underlying patterns during busy rounds even when the historical information supplied is incomplete, inaccurate, and poorly presented. The second would be the ability to reject what seems to be a coherent presentation and a logical conclusion and offer alternative hypotheses. "The facts speak for themselves" can be an erroneous simplification of a more complex underlying principle. Clinicians can always offer alternative diagnoses to explain observations, thus facts alone cannot lead to knowledge of underlying diseases. This was pointed out by Reginald Jones as "Crabtrees bludgeon"; the clinical tendency to match facts with personal unifying theories of causation. The expert history taker would guard against this type of error by testing the validity of diagnoses offered by competing hypotheses.

What should be the format of the presentation of history?

The initial opening statement should clearly state the premorbid conditions, ADL of the patient, family background, key decision makers, and socioeconomic status of the patient.

The historical statement should proceed in chronological/geographical sequence and specify the source and validity of information at each point in time/space. The narration can be a mix of symptoms and tests as most ICU patients would have passed through multiple healthcare givers. It is very difficult to capture and succinctly summarize historical details of patients with prolonged hospitalizations and multiple ICU readmissions. At each stage of the presentation, the working hypotheses with supporting evidence should be presented. Alternative diagnoses should be presented and are essential parts of "critical history taking." Symptoms in history that are significant and for which no hypotheses can be framed should be presented as they may be useful in the future. A suggested template for history-taking is shown in **Table 2**.

TABLE 2: A template for history-taking.

Headings	*Example*	*Rationale*
1. Background history	A 72-year-old is the primary caregiver for his wife who has severe arthritis. Children are abroad. He has diabetes on insulin with nonoliguric renal failure and COPD. He can walk daily for 2 km. He has insurance coverage for three lakhs and some social support from two nephews	• Age, comorbidities, and ADL have been identified • Social stressors—wife's condition, lack of immediate family support, and insurance levels have been picked up. These must be explored further in detailed interviews • The background is presented first as the patient's history must be understood in the context of the underlying conditions
2. Presenting a complaint	Chest pain for 2 days and difficulty breathing for 1 day	Mentioning in chronological order may help in finding the sequence of symptom development and that may guide us in arriving diagnosis

Contd...

Contd...

Headings	***Example***	***Rationale***
3. Indication for ICU admission	Found to have a saturation of 90% on 10 L oxygen and increasingly tachypneic. US did not show B-lines and ECG and troponin were normal. ABG did not show significant CO_2 retention	Organ failure is a common indication for ICU admission and how it was picked up should be stated and checked for validity. It is more efficient to mention the results of diagnostic interventions done prior to ICU as part of history
4. History of presenting complaint	The patient had chest pain for the past 2 days, predominantly on the right side, it was exacerbated by coughing, but there was no sneezing or nasal stuffiness. The chest pain was constant, and not episodic, not associated with sweating or palpitation, and not referred to other parts. Initially, there was only a dry cough and on the second day, there was whitish sputum. There was also wheezing on the second day. The pain was not worse on walking	An attempt is being made to look for etiologies of the cough and differentiate it from cardiac and respiratory causes of chest pain
4. (a) Course prior to hospitalization	On the first day, the patient consulted his physician who increased his MDIs and prescribed azithromycin. The patient was able to sleep that night. However, on the second night, he felt worse and could not sleep due to the cough. In the morning, he was tired and unable to walk to the bathroom and had to be brought to emergency with nursing assistance	The tempo of the illness should be portrayed. Prior information about antibiotic use is essential as therapy must be modified accordingly
4. (b) Course in emergency	In the emergency room, he was hemodynamically stable. ECG and troponin were noncontributory. Screening ECHO was uninformative due to poor ECHO window. The limited US of the lungs showed no B lines. ABG showed hypoxia. The SpO_2 90% on 10 L oxygen, PaO_2 55 mm Hg, and $PaCO_2$ 45 mm Hg. As RR was 45 breaths/min and due to comorbid conditions, the ICU transfer was thought appropriate. Appropriate antibiotics were instituted	The presentation of investigations rapidly is a departure from orthodoxy. However, this is standard and practical. The listener now knows that acute cardiac events have been ruled out, but RV dysfunction has not been evaluated. A detailed US examination, and further analysis of ABG is needed. Differential diagnoses have been raised and excluded without formal specification
4. (c) Course in ICU, diagnosis, and plan of management	In ICU, further complete US could not be done as patient was breathless. NIV was instituted overnight. He was tolerating NIV and RR came down from 45 to 35. He continued to be hemodynamically stable; MDI and antibiotics were continued. Physical examination was not contributory. CXR showed only feature compatible with COPD. A further US exam was planned	This is the stage when the history and examination findings are presented together. Differential diagnoses are offered along with supporting points in history and investigations. The validity of these assumptions is checked, and a plan is approved for management

(ABG: arterial blood gas; ADL: activities of daily living; COPD: chronic obstructive pulmonary disease; CXR: chest X-ray; ECG: electrocardiography; ECHO: echocardiography; ICU: intensive care unit; MDI: multiple dose inhaler; NIV: noninvasive ventilation; RR: respiratory rate; SpO_2: peripheral arterial oxygen saturation; US: ultrasonography)

The presentation of the history should conclude with a management plan, a statement of unresolved issues, and what were the stated wishes of the patient and family. The sign of skillful history-taking would be that at the end of the presentation the consultant has nothing to suggest and has enough information to meaningfully engage with family.

SUMMARY

Organ dysfunction is the common pathophysiological indication for ICU admission. The aim of history taking is to skillfully elicit information that will suggest causative etiologies. The presentation of history should be in chronological order, and should include symptoms and signs, investigations, and sociocultural background issues that are relevant. The principles of critical thinking should be applied and generation of alternative or counterfactual etiological hypotheses should be encouraged.

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Chapter 2

Bedside Etiquette in a Critically Ill Patient

Vijaya P Patil, Madhavi G Shetmahajan

> *It is unsettling how little it takes to defeat success in medicine. You come as a professional equipped with expertise and technology. You do not imagine that a mere matter of etiquette could foil you. But the social dimension turns out to be as essential as the scientific matter of how casual you should be, how formal, how reticent, how forthright. Also how apologetic, how self-confident, how money minded. In this work against sickness, we begin not with genetic or cellular interactions but with human ones.*
>
> ***—Atul Gawande***

ABSTRACT

Bedside manners or etiquette constitute the art in the practice of the science of medicine. Compassion and empathy should underlie all interactions with the patients particularly in stressful conditions such as in the intensive care. Unique to the intensive care, the patient and their relatives have a long exposure to the medical staff and medical systems in times of extreme emotional and financial distress. The demeanour and behavior of the intensive care unit (ICU) staff can leave a lasting impact on the lives and attitude of patients and their relatives. This chapter describes the importance of verbal and nonverbal communication skills through real-life examples which the readers are likely to identify with and encounter in their practice.

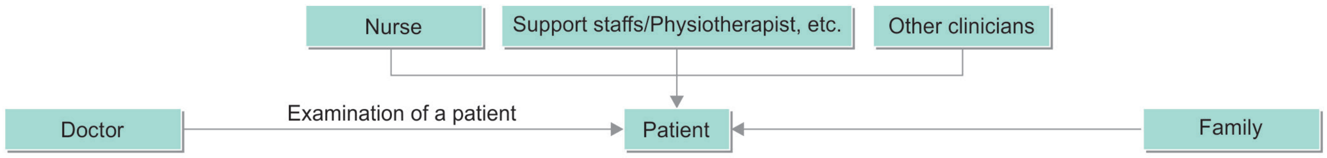
INTRODUCTION

As children, our parents and teachers impress upon us the importance of good manners. We have been taught to show respect toward the feelings and sentiments of others and show equal regard to everyone. Good manners are not just about saying words such as thank you and sorry. They include how you present yourself, how you behave, how you speak, and how well you treat others. Modesty, humility, kindness, and courtesy are essential aspects of good manners **(Flowchart 1)**.

Ethics and etiquette are branches of philosophy that advise about right and wrong. The Oxford dictionary defines ethics as moral principles that govern a person's behavior or the conduction of an activity. Etiquette is a set of rules that indicate the proper and polite way to behave in society. Unfortunately, these aspects are not formally taught during medical education but can be imbibed through observation and role modeling. Seniors play an incredibly important role by setting an example. Although knowledge and skills are essential for being a good doctor, attitude and manners are equally important. The way a doctor relates to his patient is vital. This has been recognized as an essential skill since the origins of medical science. Unfortunately, in modern times, technology has distanced the doctor from the patient. This aspect is in need of urgent revival. Hippocrates had said this in 400 BC "The patient though conscious that his condition is perilous, may recover his health simply through his contentment with the goodness of his physician." Though modern doctors may question the veracity of this claim, it cannot be doubted that a doctor's demeanor can promote the patient's mental wellbeing, if not always the physical cure.

An intensive care unit (ICU) is an intense and busy environment with a multitude of medical and paramedical staff. The interactions with the patient and between the staff is often complex and result oriented. Most physicians focus and spend most of their time on clinical management. In addition, many patients are

Flowchart 1: Medical etiquette.



Before entering the patient room/cubicle:

1. Appropriate dress codes
    - Presentable attire
2. Family is waiting outside ICU
    - Do not ignore and walk away
    - Greet them
    - Assure them that their dear one is in a good place and will be cared for well
    - Assure them that you will come back and update them
3. If the nurse is giving patient care (curtains closed)
    - Wait until she finishes her care
    - Do not open the curtain and peep in or interrupt her care

At the patient room/cubicle:

1. Greet bedside nurse
2. If the nurse is giving patient care (e.g., delivery of medications)
    - Wait until she finishes her care
    - If a nurse is speaking to the patient/family at the bedside—wait till she finishes
3. For conscious patients—
    - Introduce yourself and explain your role
    - Introduce your team and greet them
    - Ask open-ended questions about their complaints and their needs
    - Listen attentively, make eye contact
    - Take permission for undressing and clinical examination
    - Take permission for bedside teaching
    - Reassure and answer all questions
    - Take permission before leaving
4. The patient may be unconscious—it is a good gesture to still say “hello”
5. Ask the bedside nurse if she has any observations, questions, or concerns regarding patient care
6. Family is with the patient
    - Greet family members
    - Encourage them to ask questions or express their feelings
    - Gently ask permission to examine the patient or ask them politely to wait outside till you finish the examination
7. If another clinician is examining the patient, wait for them to finish
8. Do not argue or discuss at the bedside
9. Many members are present in the team
    - Avoid simultaneous multiple tasks
    - Avoid pointing out mistakes of any team member at bedside

Leaving the patient room/cubicle:

1. Ask the patient if he/she has any questions
2. Explain to them about the further course of plan of action
3. Ask the bedside nurse if she has anything to add or concerns or suggestions
4. Talk to family and update them about patient’s status
5. Encourage family members to express their concerns and ask questions
6. While leaving after finishing your duty, let family members/patient know who will be looking after their patient
7. Always address the patient by name
8. Show respect to deceased
9. Respect family emotion, spend time while you counsel

either deeply sedated on ventilator or may have altered level of consciousness in the early stages of the ICU stay, which precludes formation of the normal doctor–patient relationship. The patient's identity becomes linked with his pathology in the doctor's mind, and the progress of the disease process the defining factor in the interaction at the patient's bedside. A patient's consciousness becomes a sign to be monitored. The fact that he/she is regaining emotional competence is unwittingly forgotten. In this state of mental fragility and confusion, reassurance or even a simple acknowledgment from the doctors can help the patient adjust to their unfamiliar and uncomfortable environment.

Fear and worry are expected in all patients admitted to the ICU. Quiet confidence and empathy from treating doctor is important in calming the patient and the relatives and building a trusting relationship.

Although each patient–doctor interaction is unique, there are certain situations that are common or unusually complex. Let us look at common shortcomings regarding bedside etiquette from a few real-life scenarios.

Studies have shown that patients are more likely to be honest and satisfied when physicians are compassionate and not just competent. Physicians who adopt a warm, friendly, and reassuring manner are more effective than those who are formal and do not offer reassurance.

Effect of attire: Physicians' appearance is closely monitored and patients still expect our doctors to appear neat, without much adornment. Thus, a doctor's presentation establishes trust and authority in the doctor–patient relationship.

CASE SCENARIOS

1. *A 22-year-old young boy involved in motor bike accident, presented to emergency room with right tibia compound fracture, blunt abdominal trauma, and extensive laceration on back. He is shifted to your ICU after a computed tomography (CT) scan head and abdomen for stabilization before taking to operating room (OR) for fracture fixation.*

   While entering ICU you see parents waiting at entrance, mother is distressed and crying. Do not ignore them and walk away. Introduce yourself to them and gently reassure them that you understand their fear for their young son and that their son is in best place where he will be well cared for. Tell them that you will examine him and come back and talk to them in detail. If you see many relatives, you can gently tell them to follow hospital rules but do not be rude.

   On reaching the patient's cubicle, you find the neurologist examining the patient with the nurse in attendance. You should wait for him to finish his examination. Peeping in to see if he has finished or calling the nurse to discuss/ask your queries should be avoided. Once neurologist has finished with his examination, you can ask for his findings/management plan and then with his permission proceed with your examination.

   The patient is awake, anxious, and in extreme pain due to fracture and lacerations. Introduce yourself and explain your role. Acknowledge that you understand that he is in pain and reassure him that pain killers will be given soon. Take patient's permission to undress him and perform an examination. It is very likely that the patient has been seen by multiple doctors (e.g., emergency physician, orthopedic surgeon, general surgeon, etc.). Patients may become more anxious or irritable with repeated questioning, particularly if their distressing symptoms have not been treated. Tell him that his parents are waiting outside and you will allow them to visit him soon. Give him pain killers and explain about further plan. While leaving, introduce him to your team and reassure them that one of them will always be there to attend to his needs.

   After finishing your evaluation, call the parents and tell them about your further plans. Assure them that you will monitor their son's progress and keep them informed of any significant events. You can accompany parents to bedside and ask if they have any questions. Introduce your on-duty colleague if you are leaving.
2. *A 3-year-old child, case of acute myeloid leukemia on treatment, presents with neutropenic sepsis and develops severe multiorgan dysfunction. According to your audit, patients with prolonged neutropenia lasting >3 weeks during the induction phase and those who develop septic shock have a mortality risk >90%. You believe that in view of poor prognosis, intensive care should be limited whereas the young oncology consultant wants to continue aggressive management. There is a heated argument at the bedside regarding your individual experiences and evidence.*

The child's parents later accuse you of being unfair to their child and not treating him adequately.

Learning points: Never let your patients hear your complaint about your coworkers or other patient or their families, or use body language that shows contempt toward your colleague. Disagreements should be debated out in the office rather than at the bedside, be it with your resident, bedside nurse, or a fellow consultant. The patient can then be counseled about the options and the relative chances of success and cost of treatment.

3. *A 74-year-old well informed gentleman undergoes a Whipple procedure and requires postoperative ventilation. He is extubated the next morning. The operating surgeon has not yet spoken to the patient. Your registrar reports the patient's condition to be stable and you quickly start moving to the next patient without interacting with the patient. The patient calls out to you and repeatedly asks for the details of the surgery and his prognosis. You express displeasure at being called out and casually answer that everything is all right and leave.*

   The patient is heard complaining to his relatives that the doctors in ICU are careless and rude.

   Learning points: Always greet patients and spend a little time at patients' side even if no active interventions are needed. This shows that you care for your patient and reassures the patient that he is well looked after. Listen when patients talk. If their questions concern another specialty, politely explain that their queries require another expert to opine and that they will be contacted to inform them about the patients concerns.
4. *A 68-year-old lady is admitted with biliary sepsis and septic encephalopathy. She is verbally abusive to all her caregivers. The patient's daughter is upset and is extremely apologetic about her mother's behavior. She is seen crying in waiting area.*

   Learning points: Patient care should extend to the patient's family. They should be counseled about abnormal behavior patterns as a result of the disease. Fear of losing their loved one, guilt, and feeling of helplessness are common in family members. These should be recognized and addressed appropriately. This helps in establishing a healthy relationship with patients and their families. When talking to the relatives, a humane rather than a dry technical approach may encourage them to open up about their feelings and concerns.

   The patient–clinician relationship has both emotional and informational components. Emotional care includes mutual trust, empathy, respect, genuineness, acceptance, and warmth. Cognitive care includes information gathering, sharing medical information, patient education, and expectation management.
5. *A 62-year-old lady with metastatic Ca ovary planned for chemotherapy presents with respiratory distress. Acute coronary syndrome with cardiac failure is the most likely diagnosis based on the electrocardiography (ECG) and echocardiography (ECHO) findings. When the husband is interviewed for details regarding her cardiac history, he goes into a detailed description of her abdominal symptoms and cancer-related concerns. The doctor is rushed as he/she has a meeting to attend, so he/she interrupts repeatedly and curtly asks the husband to answer what he is asked. The husband gets agitated and says that the doctor is unnecessarily delaying her chemotherapy.*

   Learning points: Spare time when talking to the patient/relative. If you have limited time, you could say that you have 15 minutes to talk before an important meeting and you would continue the conversation later, if left unfinished. Talking is cathartic and simply listening to patients helps to ease the worry and pain in the patients. Making eye contact during the conversation conveys that you are paying attention.

   Learning to listen well can improve both work-related relationships and personal ones. People feel much more at ease when they feel heard, no matter what the context.
6. *A young college student is admitted with severe respiratory distress and is on noninvasive ventilation (NIV). On a particular day in ICU, there are 4 deaths within 2 hours. During the handover round, the resident doctor makes a comment that "they are dropping down like flies."*

   The student's sister is very upset and confronts the registrars accusing them of considering patients' life as a joke and that they are not serious in their management. The registrar angrily recounts how hard he/she has been working all night and not had a minute's rest. The argument deteriorates to a shouting match.

Learning points: ICU is a stressful environment both for doctors and patients and their relatives. Sometimes casual comments made by doctors may appear insensitive to patients because of the differing perspectives. While disease and death is a part of work life for intensive care doctors, it is the object of intense fear and stress for the patient. So doctors have to be mindful to keep light banter to a minimum at the bedside as it can be misconstrued by the patient.

Also apologizing sincerely when a mistake is made is the most appropriate response and prevents escalation of ill feelings.

7. *A 72-year-old gentleman is admitted to ICU with diabetic ketoacidosis (DKA) and diabetic foot. The intensivist orders diabetology, surgery, nephrology, and cardiology consults for ECG and blood chemistry abnormalities. After 3 days, when the patient gets the bill, he questions the visits by the doctors. He loudly accuses the intensivist of "looting" him.*

   Learning points: Always inform the patient about the various problems and the need for specialist opinions for the treatment. As a doctor, it is always good practice to introduce oneself and explain one's role in the care to the patient so that the patient is aware of the consultation. Often many medical and paramedical staff visit the patient and the patient may not remember the visit. This may lead to a feeling of being inappropriately charged.

   Also if the patient uses abusive language, politely ask the patient to refrain from using such language. Invite them to discuss their grievances in a civil manner and if required, refer them to the public relations/grievances department.

8. *A patient is admitted to the ICU after being discharged from another hospital against medical advice. She has postpartum hemorrhage and is hypotensive on admission. She is resuscitated and is then found to have retained products of conception for which she undergoes surgery. The relatives blame the previous hospital for negligence and want you to confirm it.*

   Learning points: In this case, it would be prudent to not comment on the previous medical care, particularly when the details are not available and the patient is discharged against medical advice. The communication is best restricted to the current condition and its management. One should avoid criticizing and condemning other doctors.

9. *A 20-year-old girl presents to casualty with symptoms of acute gastroenteritis. However, she remains hypotensive despite adequate fluid resuscitation and the doctor wants to rule out a cardiac cause. Wanting to perform a 2D ECHO, the male doctor tells her that he will be doing a heart scan and hurriedly draws the curtain and proceeds with the scan. The patient's mother is watching from across the partially open curtain. She is disturbed to a see a male doctor disrobing her daughter. She feels it is inappropriate when her daughter just has diarrhea.*

   Learning points: Always ask the patient's permission before exposing sensitive parts of the body. Explain the need for the procedure and what is expected from the patient to conduct the procedure.

   Always ensure privacy. Also when examining female patients, it is advisable to have a female attendant in the room.

10. *A 50-year-old trauma victim dies from a road traffic accident and severe head injury. The doctor informs his wife and tells her that the "body" will be handed over to them after 2 hours required for the necessary paperwork. The lady is visibly disturbed to hear her husband referred to as "body."*

    Learning points: We frequently address a patient by the bed number or by his underlying pathology rather than by name. We often hear statements like "the pancreatitis guy in bed number 112 has grown XYZ bug from his blood." We forget that bed number actually belongs to a person who has an individual identity, is a friend, spouse, or parent. It is quite disturbing to hear a loved one being addressed as a body. While addressing the family, always use the name of the patient, for example, "Raj's condition is improving and he had been without fever for last 24 hours," "You can take Raj after the necessary paper work," etc.

11. *On the ICU rounds, a 70-year-old man, recovering from an exacerbation of chronic obstructive pulmonary disease (COPD) complains that he had been asking for bed pan for last 10 minutes. The doctor continues the examination and informs him that he will soon be discharged to the ward. The doctor says that all staff members are busy and he will be attended soon and leaves.*

Learning points: This can be a frequent scenario in a busy ICU with inadequate manpower where attending to more serious patients and important clinical activities are given priority over more mundane activities like giving a bed pan. But, for the patient, this is as distressing and disturbing as any illness and creates a feeling of discontent against the hospital staff. In such cases, the better approach would be apologize for the delay and wait till he is attended to.

Do's

1. Greet the patient and relative.
2. Introduce yourself—name and role.
3. Ensure privacy—curtains, allow/disallow relatives in ICU during rounds according to unit policy.
4. Start with an open-ended question regarding the patients' health, e.g., how are you feeling/are you feeling better? If patient in obvious distress, ask what is troubling them?
5. Listen to the patient/relative and address their concern. Do not ignore/disregard their complaints.
6. Ask permission/inform patient before examining or exposing covered parts of the body, for example, can I lift your shirt to examine your lungs?
7. You can discuss findings/diagnosis with the team in the patients hearing but any arguments/disagreements should be avoided. Do not disparage other doctors at the bedside of the patient.
8. Reassure the patient/discuss further plan with the patient in brief before leaving the patient's bedside. If plan is not clear or if you are in a junior position, defer the decision to your senior.

Don'ts

1. Be hurried/casual in your behavior toward the patient.
2. Discuss bad prognosis casually. Hold a formal discussion with patients' relatives in the counseling room.
3. Discuss financial issues with patients' relatives at the bedside.
4. Disown responsibility of any aspect of care. Inform politely that the concerned doctor will be called at the earliest. If the problem is severe, stay with the patient till the specialist arrives.
5. Discuss patients' pathology or consult your colleague in the corridor or common areas, where other relatives may overhear.
6. Shout or point out mistakes of residents or nurse at bedside.

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Chapter

3

General Examination of a Critically Ill Patient

Bhuvana Krishna

"The art of the practice of medicine is to be learned only by experience, tis not an inheritance; it cannot be revealed. Learn to see, learn to hear, learn to feel, learn to smell and know that by practice alone can you become expert."

—Osler (1919)

ABSTRACT

General examination of a critically ill patient plays a very important part in the diagnosis of the underlying disease etiology and the associated systemic complications. A systematic format for examination from head to toe needs to be followed just like in the examination of patients admitted to the ward. The general examination format for a stable ward patient cannot be applied to a critically ill patient. This chapter outlines a systematic approach that can be adopted for a comprehensive general examination of a critically ill patient.

INTRODUCTION

The art of medicine revolves around the concept of a doctor solving a diagnostic mystery of a patient. The doctor uncovers clues from a detailed history and physical examination and performs ancillary tests to arrive at the right diagnosis. There are no chapters or sections in any textbook detailing a uniform and systematic examination of a critically ill patient. It is important to take note of the stark differences between the physical examination of a critically ill patient as compared to a stable patient in the outpatient or inpatient departments. First, the critical condition of the patient may obscure the many signs of the primary disease making it difficult to arrive at the right diagnosis through a general physical examination. Second, a rapid and thorough physical examination is very important and often lifesaving in critically ill patients, and the focus of the attending physician is often directed at resuscitation and stabilization of the critical condition. Unfortunately, the complete physical examination is often disjointed because of the number of tubes, catheters, invasive monitoring devices, dressings or the general tension, and activity that prevails around the critically ill patient. Lastly, the general examination should always include not only the body parts of the patient but also the various tubes, devices, gadgets, and ventilator attached along with the information they provide.

Little importance is given to a comprehensive examination of critically ill patients. The false assurance that the monitors and investigations will give the necessary information is a common reason. Apart from the diagnostic utility of physical examination, the human touch provides a sense of involvement and bonding to the patient and the family.

This chapter aims to provide a format for a systematic general examination of a critically ill patient, to avoid omissions.

CASE SCENARIO

A 65-year-old male patient had presented to the emergency department with lethargy for the last 5 days, poor oral intake, and increased drowsiness since last 24 hours with one episode of seizure. He is a known case of hypertension on regular medications. There is no history suggestive of substances of abuse. In the emergency department, he was intubated for low level of sensorium of Glasgow Coma Scale (GCS) of 6/15. His noncontrast computed tomography (CT) *of the brain showed no abnormalities other than an age-related atrophy and chronic ischemic changes.*

GENERAL PRINCIPLES

The general physical examination of a critically ill patient should start with the preparation of yourself and the patient. After taking a detailed history from available sources—family members, attendants, charts, old records, and if possible, from the patient—you proceed to examination. Remove all ornaments from hands, fold sleeves, and after ensuring hand hygiene, move to the bedside of the patient. The right side of the patient as always recommended may not always be feasible in the critically ill patient with many devices hindering our proximity to the patient. The examination area should be well lit, so as to quickly note fine details and signs of the patient, e.g., yellowish discoloration of the skin and eyes. Obtain the languages familiar to the patient from the bedside nurse, who would have already been in communication with the patient and family. Quickly assess the situation prevailing around the patient to help understand and prioritize your examination. If the patient is seriously ill, focus on a rapid physical examination for resuscitation and after stabilization a detailed general physical examination can be performed. Irrespective of the sensorium, it is important to introduce yourself to the patient and explain what it is that you are going to do. It is essential to have a chaperone during examination, even if the gender of the doctor and the patient is the same. It could be the bedside nurse or another doctor, preferably the same gender as the patient. Obtain consent from the patient for exposure of the body and for examining. Tactfully request the family members to be seated in a quiet area (waiting area/counseling room) away from the patient, explaining to them that you will come and speak to them after a complete assessment. Draw the curtains around the patient, in the presence of the chaperone, expose the body parts completely for examination, keeping in mind the sensitivity of the situation at the same time being professional. Body parts can be exposed in sections during examination to avoid unnecessary prolonged exposure to cold and cause embarrassment. For example, cover the chest and abdomen after examination with a blanket or gown, then move to the pelvis and lower limbs for examination. Very often in the intensive care unit (ICU), privacy of the patient is disregarded or disrupted because the patient may be in an unconscious state or the general traffic around the bed by the attending team of doctors and nurses. Irrespective of the intensity of treatment, patient privacy and modesty should be always ensured.

The systematic examination should be aimed at finding clues in signs to confirm or refute the differential diagnosis made after obtaining the history. The basic components of the physical examination—inspection, palpation, percussion, and auscultation should be followed where applicable. The point-of-care ultrasound (POCUS), an addition to these basic steps, should be incorporated where available and feasible for completion of the study.

VITAL SIGNS ASSESSMENT

The assessment of vital signs is the first step in physical examination as soon as you reach the bed space of the patient, even before taking the history or takeover details. The ever preached and practiced steps of A-B-C should be checked and maintained throughout the examination period. This examination should not take >10 seconds. Advanced cardiac life support steps need to be initiated wherever the A-B-C components are absent.

A—Airway patency: The patient may be shifted to the emergency or to the ICU, with or without supplemental oxygen or endotracheal tube; patency of the airway is the first vital sign check. Threatened airway or obstructed airway can be recognized by an astute physician in a nonintubated patient by either a noisy/stertorous breathing, gurgling sounds, or stridor. In an intubated patient, it can be recognized by high peak airway pressures with low minute ventilation on the ventilator. Tube obstruction can be confirmed by the difficulty in passing a suction catheter. The endotracheal tube could be malpositioned, then an immediate check laryngoscopy will be necessary. There will be signs of impending respiratory failure also—diaphoresis, obtunded sensorium, cyanosis, paradoxical or see-saw movements of the chest, and abdomen and accessory muscle use.

Expiratory flow in a nonintubated patient can be checked by keeping the volar aspect of the forearm or an ungloved physician hand against the mouth and nose. If expiratory flow is detected, it rules out complete obstruction.

Management: First, any foreign bodies need to be manually removed after inspection of the airway. Opening of the airway manually can be achieved by a head tilt chin lift, which will lift the tongue, soft palate, and the epiglottis away from the glottic opening. The other method is the jaw thrust method, which can be employed in cases of cervical spine injury or instability. An oropharyngeal or nasopharyngeal airway can be used to keep the airway patent until a definite airway can be secured after endotracheal intubation. In case of any obstruction to the endotracheal tube, clearing it with suction or insertion of new tube may be necessary.

B—Breathing: It can be checked by both inspection and palpation of chest rise. Symmetrical chest rise can also be noticed. Absent breathing or abnormal breathing may be noticed in the form of agonal gasps or gasping, grunting, or apneustic breathing.

Management: If there is absent or abnormal breathing despite patent airway, assisted breaths with an Ambu bag or ventilator is to be instituted.

C—Circulation: It is checked by palpating the central pulses such as the carotid or femoral with three fingers. The presence of a pulse just excludes a cardiac arrest state and does not confirm adequate tissue perfusion. Checking the adequacy of radial pulse volume may not always confirm adequate circulation, e.g., in the case of sepsis, bounding pulses can exist with hypotension. The rhythm and rate of the pulse can be checked with a hand on the pulse while the monitors are being connected. Blood pressure should be measured by either a noninvasive or invasive method where available. Adequacy of tissue perfusion is assessed by checking capillary refill time (CRT). CRT is the time taken for color to return to an external capillary bed after pressure is applied. Typically, pressure is applied to the nail bed of a finger until it turns white. After releasing the pressure, the color should return in <3 seconds if circulation is normal. If it takes >3 seconds, then tissue perfusion is poor.

Management: Circulation needs to be optimized by firstly determining the cause for the shock—septic, cardiogenic, obstructive, or hypovolemic shock. The approach to shock will be dealt in detail in subsequent chapters.

Once the A-B-C adequacy is ensured, the vital signs are examined in detail. The vital signs will give an idea of the patient's critical condition, which can be attended to before a detailed structured examination can begin.

- *Pulse:* Rate, rhythm, and if all peripheral pulses are felt—check bilateral radial, femoral, posterior tibial, and dorsalis pedis arteries. Absent pulses may indicate peripheral vascular disease or arterial thrombosis. Check the electrocardiogram (ECG) reading from the monitor for any abnormalities. Tachycardias and bradycardias will need immediate attention as per the advanced life care support (ACLS) protocols.
- *Blood pressure:* Check whether invasive or noninvasive method of measurement and if on vasopressors, the dosage should be noted. Invasive blood pressure readings from which artery should be noted—radial, femoral, dorsalis pedis, or posterior tibial are some sites for invasive arterial line placement. The position of the transducer if correctly placed at the phlebostatic axis (4th intercostal space in the midaxillary line) and when was it last zeroed is also important. The interpretation of the arterial waveform with respect to the shape and position of dicrotic notch can give in-depth understanding of the arterial system mechanics. The fast flush test should be done to check if the waveform is adequately damped. Arterial line tracing for any swing will indicate the intravascular volume status. Complications due to invasive arterial line position should also be noted—in the form of discoloration of distal limb, swelling, decreased temperature, and blistering or absent pulses.
- *Saturation:* The oxygen saturation and on how much fraction of inspired oxygen (FiO_2) is noted along with oxygen delivery device used. The partial pressure of oxygen (PaO_2)/FiO_2 ratio and the positive end-expiratory pressure (PEEP) if on the ventilator will give an idea of the severity of the lung disease. If the saturation is not getting detected (a common scenario), the reasons could be cold peripheries or painted nails.
- *Respiratory rate:* A normal respiratory rate is 12–20 breaths/min, but patients may need higher rates on the ventilator if he is paralyzed and has metabolic acidosis to optimize the pH. A higher rate in a conscious patient will need evaluation for causes such as hypoxia, shock, metabolic acidosis, pain, or anxiety.

Once the patient has been assessed for impending deterioration and the condition has been stabilized, a more detailed and structured general physical examination is done.

FIRST IMPRESSION

The first impression or the end-of-bed assessment or end of bed-o-gram is a quick assessment of the patient standing at the bed end, before proceeding to a complete physical examination. A quick visual scan of the patient and the surroundings can give a lot of information **(Fig. 1)**. How is the patient lying in bed—calm, comfortable, in distress, or restless? Is the patient on oxygen or noninvasive ventilation (NIV) support or is intubated? What are the vitals on the monitors? Are there infusions connected to the patient? Are there any drains, catheters, or other devices? This quick assessment not taking >60 seconds will give a general idea of the critical condition of the patient and help channelize and focus our organ system of examination. An astute and experienced physician can generate differential diagnosis with just the end-of-bed-assessment. A detailed head-to-toe examination should be performed **(Table 1)** and if needed address the emergency situations that may arise during this period.

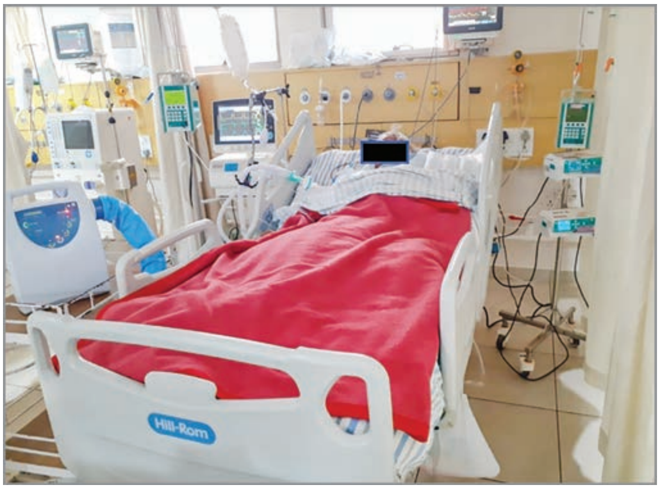


Fig. 1: Bed-o-gram.

TABLE 1: Head-to-toe examination.

General appearance	Assess the built, nutritional status (malnourished or overweight), calm or in distress, consciousness, or assess Glasgow Coma Scale or sedation scale score [e.g., Richmond Agitation Sedation Scale (RASS)]
Head	Hair for sparsity **(Table 2)**, texture, nits or lice or scalp lesions, occipital pressure sore, ear discharge—blood/fluid/pus
Eyes	• Pallor, jaundice, subconjunctival suffusion/bleed, exposure keratitis • Pupillary reaction to light, conjugate palsies • Periorbital edema, exophthalmos, ptosis
Face	Facial symmetry, inspect the ears for blood/cerebrospinal fluid/pus if indicated, inspect the nares for blood/discharge, oropharynx for bleeding, breath odor **(Table 3)**, ulcers, loose teeth, and if the endotracheal tube is fixed appropriately
Skin	Color for pallor/jaundice, lesions (rash/petechiae/ecchymosis)—diffuse or localized lesions, temperature, dryness, skin turgor for dehydration and for edema, cyanosis/poor capillary refill time/mottling **(Figs. 2 to 6)**
Neck	Jugular venous pulsations, lymph node examination, swelling (retropharyngeal abscess), cysts
Chest	Movements of the chest, shape of chest, symmetry, subcutaneous air, skin, incisions or dressings, gynecomastia (chronic liver disease/drug induced), breast lumps/nipple abnormalities/skin changes
Abdomen	Distension, skin, incisions or dressings **(Figs. 7 and 8)**
Pelvis	Swellings in hernial orifices/groin, testicular swelling, skin ulcers on the genitalia, redness, purulent discharges, growths
Upper limbs	Symmetry, deformities, edema (lymphedema/deep vein thrombosis/superficial thrombophlebitis), hand for temperature, sweating, perfusion. Nails for abnormalities such as clubbing, splinter hemorrhages, platynychia, arteriovenous fistulas
Lower limb	Edema—unilateral (deep vein thrombosis/cellulitis/lymphedema) or bilateral, skin color changes (discoloration due to vascular insufficiency), venous ulcers or nonhealing ulcers of foot, gangrene, or any signs of amputated digits **(Fig. 9)**
Examination of back	• Examination of back for spine abnormalities, skin lesions—pressure sores/rash/ecchymosis, any gluteal abscess, anal fistula • Rectal examination as indicated

TABLE 2: Hair and nail abnormalities in systemic diseases.

Hair abnormalities in systemic diseases	
Acute hair loss	• Acute severe illness • Other stressors—surgery, emotional stress, postpartum • Nutritional deficiency—iron, zinc, vitamin A • Crash dieting • Drugs—anticonvulsants, ACE inhibitors, antineoplastic drugs
Chronic hair loss	• Malnutrition, malabsorption • Chronic renal or liver diseases • Thyroid illness—hypothyroid illness can cause loss of outer one-third of eyebrow (Hertoghe sign) • Autoimmune conditions—systemic lupus erythematosus
Hair shaft abnormalities	• Protein and calorie deficiency—thin and light hair • Protein deficiency—thin and fragile hair, flag sign (alternating bands of red or white) • Drugs—antineoplastic drugs • Radiation • Heavy metal ingestion
Hirsutism	• Ovarian diseases—polycystic ovarian disease • Adrenal hyperplasia • Cushing disease • Drugs—exogenous androgens
Nail abnormalities	
Clubbing	Cardiac, lung, gastrointestinal, and liver diseases
Koilonychia (spoon-shaped nails)	Iron deficiency, hyperthyroidism
Brittle nails	Hypothyroidism, diabetes, malnutrition
Melanonychia	Pigmentation of nails with melanin—pregnancy, Addison disease, AIDS, drugs
Leukonychia	Hereditary, metastatic malignancy, Hansen disease, cirrhosis **(Fig. 10)**
Splinter hemorrhages	Bacterial endocarditis, cirrhosis of liver, antiphospholipid syndrome

Note: Skin and soft tissue lesions are discussed in a separate chapter (refer to Chapter 41 for details).
(ACE: angiotensin-converting enzyme; AIDS: acquired immunodeficiency syndrome)

TABLE 3: Breath and body odor in systemic illness.

Breath and body odors	***Systemic illness***
Alcohol intoxication	Yeasty/ripe fruit/fruity sour smell
Fruity odor (acetone breath)	Ketoacidosis
Urine-like odor	Uremia
Musty or fecal odor	Liver diseases—fetor hepaticus (breath of dead)
Chemical smell	Poisoning, especially insecticide

TUBES AND DEVICES

Patients in the ICU often have multiple tubes, catheters, drains, and lines to which infusions are attached. It is equally important to note in detail each of these tubes or devices and the drugs being administered through the infusion pumps.

- *Head:*
  - External ventricular drain may be present draining cerebrospinal fluid. The position of the drain bag,

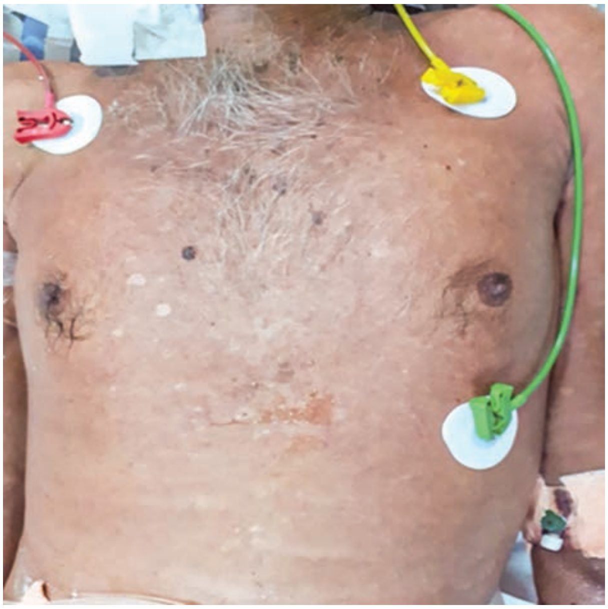


Fig. 2: Flushed trunk in a patient with sepsis.

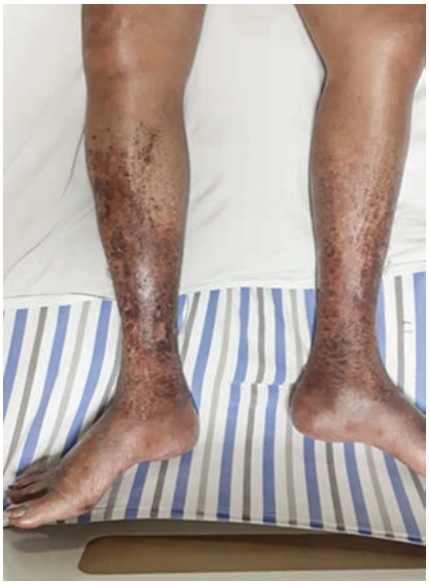


Fig. 3: Eczema over the lower limbs.

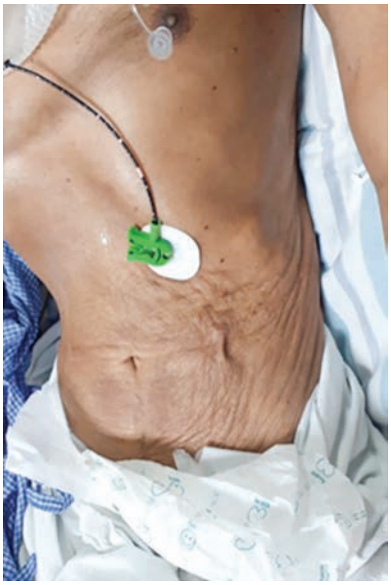


Fig. 4: Loss of skin turgor due to severe dehydration.

color of the fluid, and rate of drainage should be noted. Is there ongoing intracranial pressure monitoring, what is the pressure, is the transducer levelled correctly at the external auditory meatus (level of the Foramen of Monroe), and when was it last zeroed is also important.

- Subgaleal drains after cranial surgery may be present, note the drainage quantity and color.

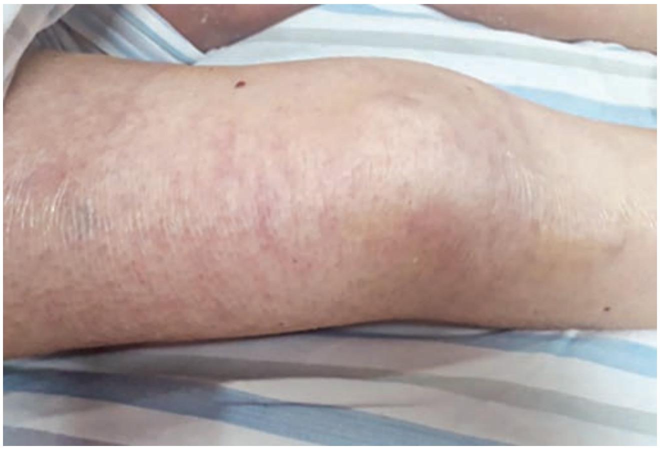


Fig. 5: Mottling of skin over knees.

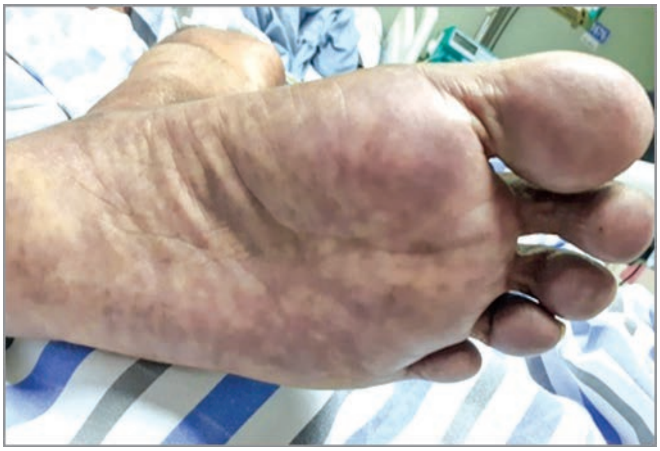


Fig. 6: Poor peripheral perfusion.

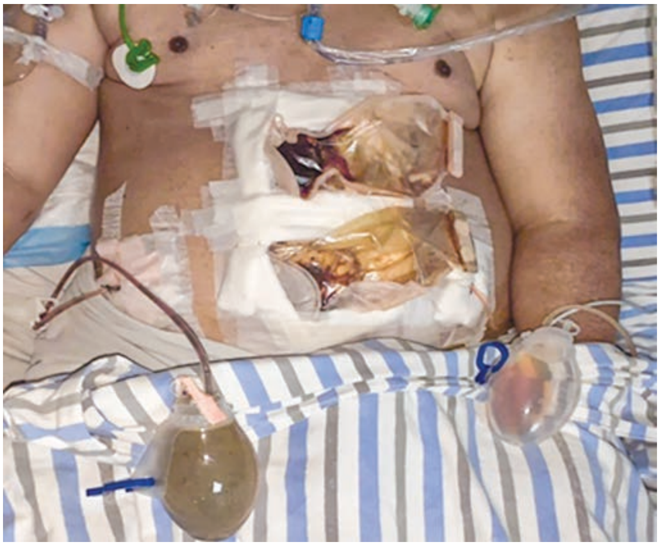


Fig. 7: Drains and colostomy bag after abdominal surgery.

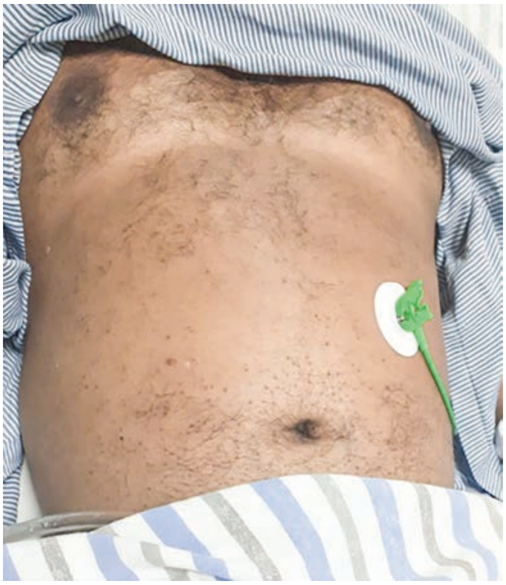


Fig. 8: Abdominal distension.

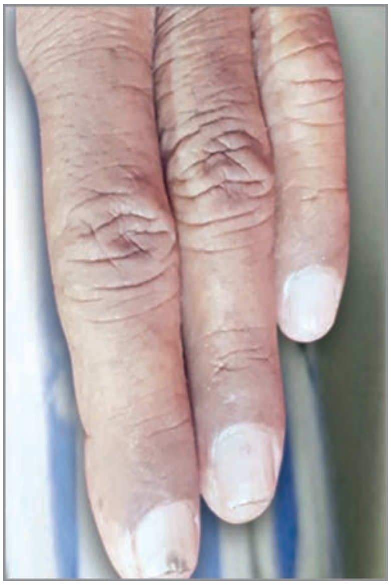


Fig. 10: Leukonychia, patient with underlying anemia.

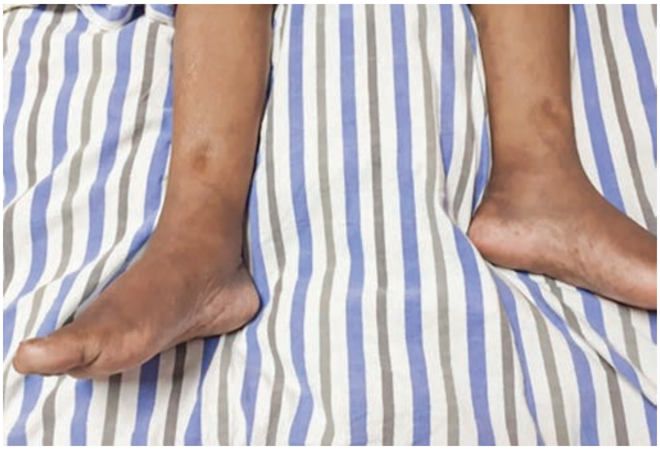


Fig. 9: Pitting pedal edema.

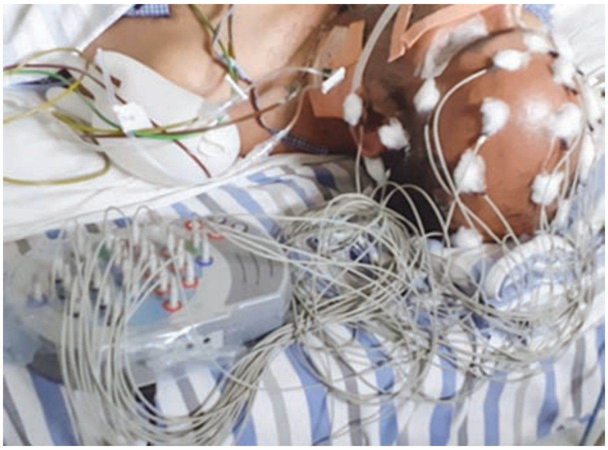


Fig. 11: Patient connected to electroencephalogram (EEG).

- Is the patient connected to continuous electroencephalogram (EEG) monitoring? **(Fig. 11)**
- Nasogastric or orogastric tube—for enteral feeding. Size (usually for adults 16–18 French), date of placement, nostril placed, position of tube, and whether position confirmed by chest X-ray. Is the patient being given enteral feeds, or is nil per oral, or is there a continuous gastric aspiration. Nasojejunal tube should be recognized from a nasogastric tube, it will give a clue to the possibility of underlying abdominal pathology.
- Endotracheal tube if present, type of tube is very important—oral or nasal, cuffed or uncuffed, material of tube, normal or reinforced tube, RAE (Ring-Adair-Elwyn tube), or double lumen tube. Size of the tube, position (teeth and lip level), intubation date, and cuff pressure (ideal is 25–30 cmH_2O or 18–22 mm Hg) to avoid aspiration and complications due to high cuff pressure. Correct position of the endotracheal tube tip as seen on chest X-ray, 5 cm ± 2 cm from the carina in adults (Goodman criteria). Other things to note of the endotracheal tube—if connected to closed suction system, is there subglottic suction and is it being used, end-tidal carbon dioxide monitoring device, and heat and moisture exchanger (HME)

or humidifier, spacers, or other extra connections. The ventilator tubing, when was it changed, ventilator mode, and the settings should be described.
- Oxygen delivery devices—if the patient is on face mask oxygen or nasal prongs, venturi mask or rebreathing/nonrebreathing mask, oxygen flows, and fit of the devices. High flow nasal oxygen therapy should be recognized and check the FiO_2 flow rates, temperature, and fit.
- Noninvasive mask ventilation devices detail should be noted—type of mask (full face, orofacial, helmet, or nasal), size of mask, mask fit, expiratory vents, and to what ventilator device—portable ventilator or ICU ventilator, the mode, and settings. It is also important to mention if patient is comfortable on NIV, hours/days on NIV, and give the HACOR score.
- If airways are placed—nasopharyngeal or oropharyngeal airways—the size should be noted.

- *Neck:*
  - Tracheostomy tube, if present, size, cuff pressure, type of tube (portex, silicon, adjustable length tube, etc.). Fixing of the tracheostomy tube and the presence of inner cannula should be noted.
  - Central venous catheters in jugular or subclavian veins may be present—note the type (subcutaneous, tunneled, silver impregnated, and antibiotic coated), lumens, size, date of insertion, and the site to be inspected for redness, swelling, pus discharge, and the type of dressing (occlusive or nonocclusive, transparent or opaque, antimicrobial dressing).

    All infusions connected to the central venous catheters should be described in detail with dosage and rate of infusion **(Fig. 12)**. If the patient is on vasopressor, the infusion volume, rate in μg/kg/min, and mean arterial pressure achieved with that dosage is noted. If the patient is on sedation, the dosage and the sedation score achieved with the drug is noted. Fluids being administered should also be described.
  - Temporary pacing catheters if present, the position should be confirmed by the pacing rhythm and on chest X-ray, pacing rate, sensitivity, and amplitude. Date of insertion and dressing is also important.
  - Dialysis catheters may be inserted into the jugular or subclavian veins. It should be described similar to venous catheters—size, type, lumens, date of insertion, dressing, and inspection of the site of insertion.

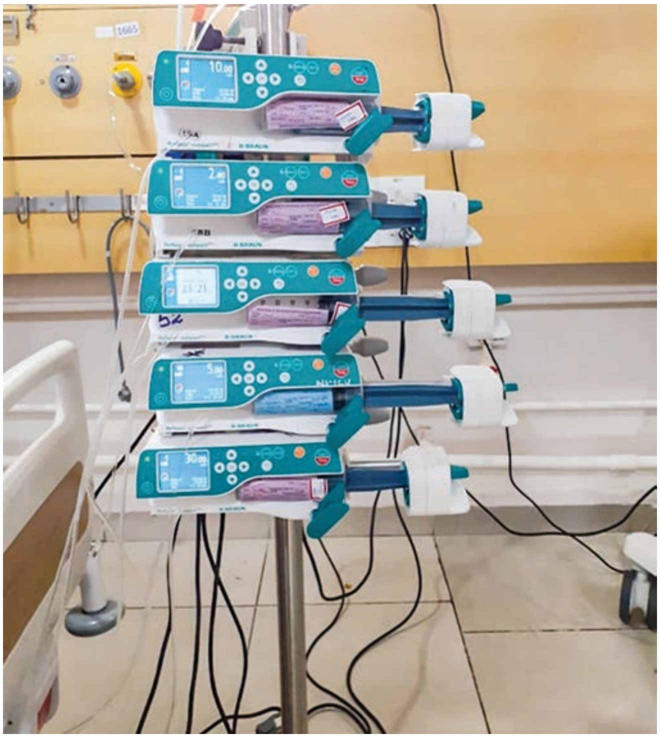


Fig. 12: Infusions.

- *Chest and abdomen:*
  - Drains should be noted for reason for insertion, draining fluid description, position, drainage system (passive or active, gravity or vacuum assisted), and dressing. It is common in ICU patients to have intercostal drains for pneumothorax or pleural effusions, abdominal drains after surgery, or for drainage of abdominal collections **(Figs. 7, 13 and 14)**.
- *Limbs:*
  - Peripheral venous cannulas, if present, the site and date of insertion along with gauge of the cannula, number of cannula, and for any signs of thrombophlebitis should be inspected. Central venous catheters in the femoral site if present should be noted in detail.
  - Spinal boards or plaster of Paris casts for immobilization or splints or skeletal traction if present will caution us for movement of patient during examination.

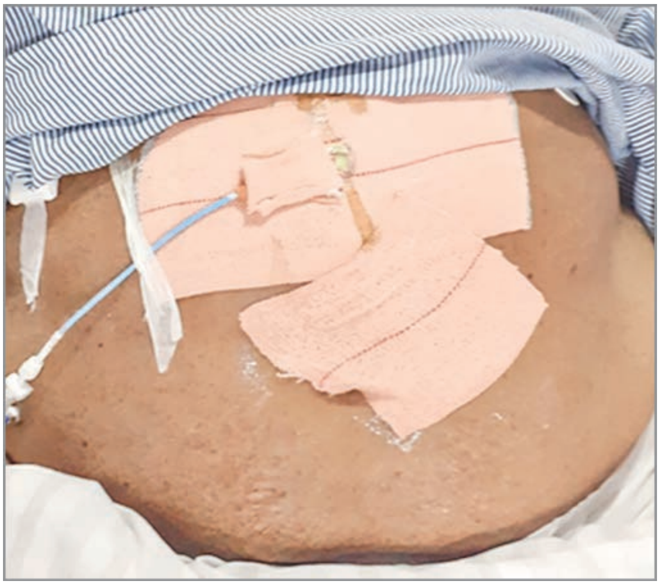


Fig. 13: Percutaneous transhepatic biliary drainage (PTBD) tube in a patient with obstructive jaundice. See Figure 14.

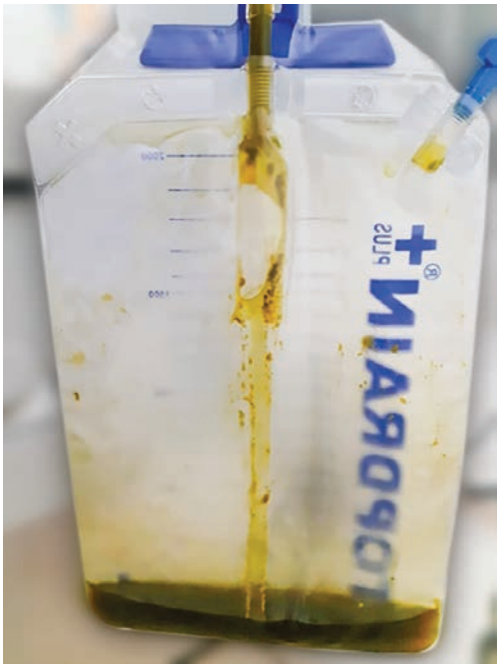
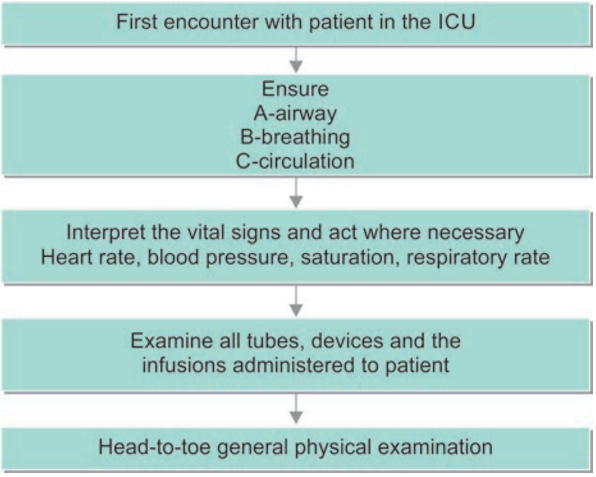


Fig. 14: Bile being drained from a percutaneous transhepatic biliary drainage (PTBD) tube.

Flowchart 1: Schema for examination of critically ill patients.


First encounter with patient in the ICU
Ensure
A-airway
B-breathing
C-circulation
Interpret the vital signs and act where necessary
Heart rate, blood pressure, saturation, respiratory rate
Examine all tubes, devices and the infusions administered to patient
Head-to-toe general physical examination

(ICU: intensive care unit)

CASE DISCUSSION

A 65-year-old male patient presented to the emergency department with lethargy for the last 5 days, poor oral intake, and increased drowsiness since last 24 hours with one episode of seizures. He is a known case of hypertension on regular medications. No use of substances of abuse. In the emergency department, he was intubated for the low level of sensorium of GCS of 6/15. His noncontrast CT of the brain showed no abnormalities other than an age-related atrophy and chronic ischemic changes.

From the history, a differential diagnosis of metabolic encephalopathy, meningoencephalitis, cerebrovascular accident such as a subdural hematoma (ruled out on CT brain) or acute stroke (less likely) can be made.

General physical examination—patient's A-B-C needs to be checked. As the patient is intubated, check the position of the endotracheal tube, after connection to the ventilator, equal chest rise, and ventilator delivering breaths, and also check for peripheral/central pulses. Proceed to examining the vitals—the heart rate, rhythm, and electrocardiographic waveforms. Note the saturation and on how much fraction of inspired oxygen, optimize where necessary. The blood pressure—noninvasive or invasive with or without supports and dosage of vasopressors being used. Once interventions to stabilize the patient are completed, the tubes and infusions should be listed, then a head-to-toe general examination should be started.

This patient on examination had a heart rate of 116 beats/min, normal sinus rhythm with all peripheral pulses that were felt. His saturation was 96% on an FiO_2 *of 0.4 and PEEP of 5. His blood pressure was 110/60 mm Hg without vasopressor support, measured through a blood pressure cuff tied on the left arm. He was afebrile, intubated with an 8.0 size cuffed portex endotracheal tube, fixed at 20 teeth*

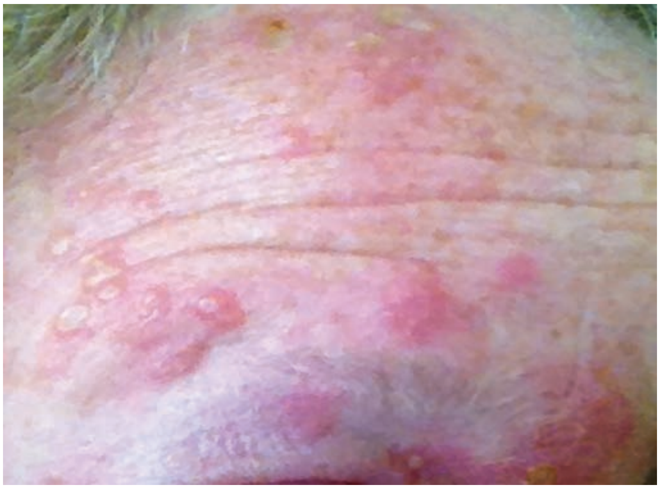


Fig. 15: Herpes zoster ophthalmicus.

level, connected to the ventilator on pressure regulated volume control mode of ventilation with an HME filter. He had a 22-gauge cannula on the right forearm, with a noradrenaline infusion that was started in the emergency room and later stopped. He had a Foley catheter for drainage of urine, connected to a drainage bag. The patient was lying calm in bed with no sedation. His GCS was—eye-opening to pain with a flexor response, with minimal pupillary light response due to bilateral cataract. On a head-to-toe examination, he was found to have a rash on his forehead, localized to the right half, with a few vesicular lesions. No other positive findings were noted. With the skin lesions, a possibility of herpes zoster ophthalmicus was made, with varicella zoster meningoencephalitis was suspected and confirmed on lumbar puncture cerebrospinal fluid analysis. After treatment with acyclovir, the patient improved in his neurological status **(Fig. 15)**.

SUMMARY

It cannot be overemphasized the importance of following a structured format for general physical examination even in critically ill patients, which should be practiced not only during the first encounter with the patient but also during the daily rounds. Practice of a thorough examination gives the necessary experience, knowledge, and prevents development of hyposkillia (lack of clinical skills) in the present and future generations of doctors **(Flowchart 1)**.

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Chapter

4

Initial Rapid Examination of a Critically Ill Patient

Shoma V Rao, Subramani Kandasamy

> *"The art of the practice of medicine is to be learned only by experience, tis not an inheritance; it cannot be revealed. Learn to see, learn to hear, learn to feel, learn to smell and know that by practice alone can you become expert."*
>
> ***—Osler (1919)***

ABSTRACT

A critically ill patient can be unstable and deteriorate very rapidly. It is therefore necessary that the management, which includes history taking and clinical examination be quick, and interventions are carried out in parallel to assessment. Certain clinical scenarios may necessitate a rapid intervention; for example, securing the airway, may not permit us to take a history or examination. In critically ill patients, the standard pathway of examination may not be possible, and a quick examination followed by a provisional diagnosis is made and a definitive diagnosis can be made as we stabilize the patient. One should understand that the degree of compensation reflects the degree of illness. This chapter will deal with this initial rapid examination of a critically ill patient.

INTRODUCTION

Clinical deterioration of any patient or individual will depend on the acuteness and the severity of the insult to the physiology of the body. For example, a myocardial ischemia/infarction can precipitate a fatal arrhythmia, and a head injury with a rapidly expanding extradural hematoma can be fatal within minutes. A patient who develops left ventricular failure following myocardial infarction may become progressively hypoxic over hours, whereas a septic patient may deteriorate slowly. The rapidity of deterioration will depend on the severity of the insult, the physiological reserve, and the speed at which supportive measures are instituted. For example, a patient who becomes septic may maintain his blood pressure with compensatory mechanisms. Special attention must be paid to the elderly, children, young adults, trauma victims, and immunocompromised as they may not present with classical symptoms.

The classical method of assessing a patient would be a thorough history taking, clinical examination, investigation, and then instituting treatment. This approach may not be feasible in emergency situations. Treatment may have to commence before arriving at a provisional/definitive diagnosis, for example, securing the airway. Treatment and assessment may have to go in parallel.

Scoring systems such as Early Warning Score (EWS) and Modified Early Warning Score (MEWS) are useful in early identification of patients.

Initial Assessment

The initial assessment has to evaluate situations that pose an immediate threat to life, namely airway, breathing, and circulation in that order. A rapid look at the patient and bed space or surroundings is very important to identify any immediate threat to life. The first examination of critically ill patients should be rapid, short, and focused. Such rapid examinations are commonly performed by the intensive care physician during every patient admission or on demand. These examinations are usually performed to identify urgent life or organ-threatening conditions and in some situations the severity of the illness that helps to plan appropriate therapeutic interventions. These are identified either by direct or compensatory signs. The severity of the illness can be gauged by the intensity of the compensatory response. The compensatory response is generally *increased respiratory drive or sympathetic when it comes to cardiovascular* manifestation. Conditions that blunt these responses have to be taken into account during the initial evaluation as will be detailed later. Any abnormality in consciousness level, airway, breathing, and circulation should be attended to in that order before proceeding to the next step. If there is more than one operator, these can go simultaneously. A patient who is not fully conscious, one whose airway is compromised, one who is not breathing, or one who does not have a palpable pulse needs immediate attention and treatment.

CASE SCENARIO

Consider a 55-year-old man with a known case of coronary heart disease presenting to the emergency department with a history of chest pain and breathlessness. His presenting respiratory rate is 35 breaths per minute and is visibly dyspneic. Pulse oximeter–derived oxygen saturation is 81% on room air. His heart rate is 130 beats per minute and his blood pressure is 88/60 mm Hg. Going by the history, one would come to a provisional diagnosis of acute myocardial infarction in cardiogenic shock. The priority here is stabilizing the airway and circulation rather than working up to diagnose myocardial infarction. This may involve noninvasive ventilation, administration of vasoactive medications via peripheral line and not intubation or awaiting central line placement for the administration of vasoactive medications. Thus, making a provisional diagnosis and initiating treatment early are more important than establishing a diagnosis.

EXAMINATION OF THE PATIENTS

The initial rapid examination of critically ill patients is done to identify life-threatening problems, impending signs of organ failures, and problems that may turn life-threatening in due course, and to resuscitate the patient to the safe zone for further detailed examination and further management. It is a good old teaching to do a rapid examination of ABCDE, airway, breathing, circulation, etc. The examination should be more systematic, rapid, limited, and focused to serve its purpose.

First Impression

Often the first impressions of the patient and bed space give clues about the disease condition. Some patients may have characteristic features, for example, cyanotic or pale gray face, a sign of impending collapse. Patients with decorticate and decerebrate postures are often not missed in acute healthcare settings. Certain breathing patterns, prolonged apnea, tachypnea with accessory muscle use, irregular rapid breathing, and Kussmaul breathing [diabetic ketoacidosis (DKA)] often give clues of diseases. One cannot forget the characteristic smell of certain conditions if you would have come across it at least once before. The smell of pesticide poisoning, for example, organophosphorus poisoning (garlic odor), the foul smell of necrotic wounds (Fournier gangrene, infected diabetic ulcers), and the smell of intoxicated patients and fruity odor in patients of DKA may give a clue to the preliminary diagnosis **(Table 1)**.

Consciousness

A sick patient can manifest anything from agitation to coma, the two extremes of sensorium. Shallow breathing, airway obstruction, and aspiration are risks and measures have to be taken to prevent these complications. It would be best to place these patients in recovery position till the airway is secured. An exception for recovery positions would be trauma victims whose cervical spine may not have been cleared.

Airway

Look, listen, and feel would be the best way to assess the airway and breathing. A patient who can speak has a clear airway. Poor sensorium with "gurgling" noise, presence of stridor, and facial, neck, and upper chest injury can compromise the patency of the airway and appropriate corrective measures, which may include an emergency tracheostomy, may have to be performed.

Breathing

One of the subtle signs of a patient who is worsening is an increased respiratory rate. Dyspnea, the use of accessory muscles of respiration, may set in at a later sign. An increased effort in breathing does not always have a primary respiratory etiology. Cardiac failure, metabolic acidosis, and any form of shock, to name a few can manifest as tachypnea. One should be aware that during

TABLE 1: Clues from first impression—rapid first-look examination.

S. No.	*Domain*	*Description*	*Comments*
1.	Posture	• Decorticate, decerebrate, opisthotonos • Bending forward holding the stomach—the pain of pancreatitis • Rolling over—colicky pain	• The characteristic postures are diagnostic of neurological problems • Patients adapt certain postures to manage their pain. Classical bedside teaching examples are patients with pain of pancreatitis and colicky pain
2.	Habitus	• Anasarca with muscle loss, ecchymosis, and dull face • Cachexic patients with severe wasting • Morbid obese	• Prolonged illness and chronic critically ill patients • Severe malnutrition and prolonged illness • OSA and COPD with respiratory failure
3.	Face	• Flushed face • Cyanosis • Pale gray face	• Septic and superior vena cava obstruction • Central cyanosis and poisoning • Impending collapse and severe circulatory failure
4.	Breathing pattern	• Irregular fast breathing • Tachypnea with accessory muscle use and anxious face with frequent random head movements • Paradoxical breathing	• Biot's and Cheyne–Stokes breathing are seen in pontine stroke and heart failures • Impending respiratory failure • Severe airway obstruction and diaphragmatic palsy
5.	Smell	• Garlic—OP • Foul smell—necrotic wounds, polymicrobial • Metallic, blood—melena, GI bleeding	• Identification of poisoning and medical conditions such as diabetic ketoacidosis, inborn error of metabolism with peculiar smell

(COPD: chronic obstructive pulmonary disease; GI: gastrointestinal; OP: organophosphate; OSA: obstructive sleep apnea)

terminal stages, the patient's respiratory system is unable to compensate, and breathing can be shallow and slow and progress to respiratory arrest. Mechanics of the chest can be worsened by pain, sedation score, etc. Following trauma, rib fractures, pneumothorax, or hemothorax can cause respiratory failure.

Checking for chest movements: Equal chest rise—

- Keep one hand over the chest and check for the movement of the hand—ensure breathing if both hands are placed on both sides, it ensures a bilateral chest raise.
- Lung ultrasound is easy and more specific for identifying ventilation.

Circulation

Heart Rate and Blood Pressure

The common presentation of circulatory decompensation is tachycardia. The etiology of tachycardia may be multifactorial. One should be aware that medications such as beta blockers, high spinal cord injury, or the presence of a permanent pacemaker may not manifest tachycardia. A biphasic response tachycardia culminating into bradycardia may be the terminal event before progressing to cardiac arrest. Abnormalities in the macrocirculatory parameter, namely blood pressure, will be delayed. The compensatory sympathetic stimulation mechanisms will attempt to maintain a mean arterial pressure that reflects the perfusion pressure. However, a narrow pulse pressure may indicate compensatory mechanisms in play.

Abnormalities in the microcirculation may have been present for a longer period before macrocirculatory abnormalities manifest. Altered sensorium, cold clammy peripheries, delayed capillary refill time, oliguria, etc., may manifest earlier before a reduction in blood pressure. It is prudent to identify these abnormalities. Shock indices (systolic shock index, diastolic shock index, and modified shock index) have been used to assess the severity of shock and may be useful in assessment **(Table 2)**.

TABLE 2: Shock indices.

Index	*Formula*	*Reference value*
Systolic shock index—useful in hypovolemic shock	Heart rate/systolic pressure	0.7–0.9
Modified shock index	Heart rate/mean arterial pressure	0.7–1.3
Diastolic shock index—predicts vasopressor requirement in septic shock and mortality	Heart rate/diastolic pressure (need more evidence)	2.0

Investigation

Point-of-care devices would be very useful in quickly diagnosing various life-threatening conditions. Point-of-care ultrasound (POCUS) is very useful in these situations. The lungs, heart, preload assessment, abdominal scan for free fluid, biliary tract, kidneys, and renal tract POCUS can largely replace a chest X-ray, but a portable X-ray should be done at the earliest available opportunity. Blood gas analysis would give a sea of information not only about oxygenation and ventilation but also perfusion (lactate and metabolic acidosis) and any life-threatening electrolyte abnormalities (hyperkalemia) or causes for low sensorium (sodium), etc. A venous sample would be good enough to diagnose metabolic and electrolyte abnormalities. Blood sugar, serum creatinine, and serum bilirubin can also be incorporated into the blood gas machine. The above with a complete blood count can be sent as resuscitation and stabilization is in progress. Other tests such as urine routine, blood culture, and sputum/endotracheal tube aspirate should be sent at the earliest depending on the clinical picture.

Monitoring

If available, a multiparameter monitoring should be instituted. A pulse oximeter would be a good tool to use.

Once the patient is stabilized, further evaluation should proceed including history, which may be obtained from the patient's relative and the treating unit if the patient is already an inpatient. Reviewing documents such as vital signs chart, fluid balance charts, medication charts, and daily progress charts, which include nurses notes will also provide more information.

Safe transfer to a monitored environment, namely a high dependency unit or an intensive care unit (ICU) or to operation theater (e.g., bleeding) should be arranged at the earliest.

GENERAL PHYSICAL EXAMINATION

Identification of signs of organ failures and life-threatening signs that require urgent interventions. A quick head-to-toe examination of critically ill patients who are recently admitted to the ICU or emergency room and a focused-on-demand examination of critically ill patients when there is new worsening is commonly performed by the intensive care physician to identify the problem that requires urgent interventions. The examination should be tailored to only look for life- or organ-threatening signs later followed by a detailed examination **(Figures 1 to 5 and Table 3)**. Refer to Sections 2, 3, and 4 for a detailed examination of organ systems.

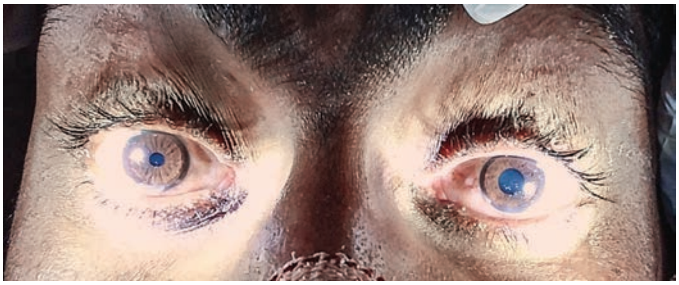


Fig. 1: Left dilated pupil in a patient with raised intracranial pressure and brain herniation.

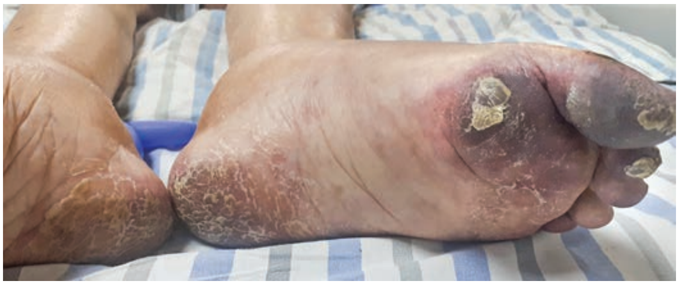
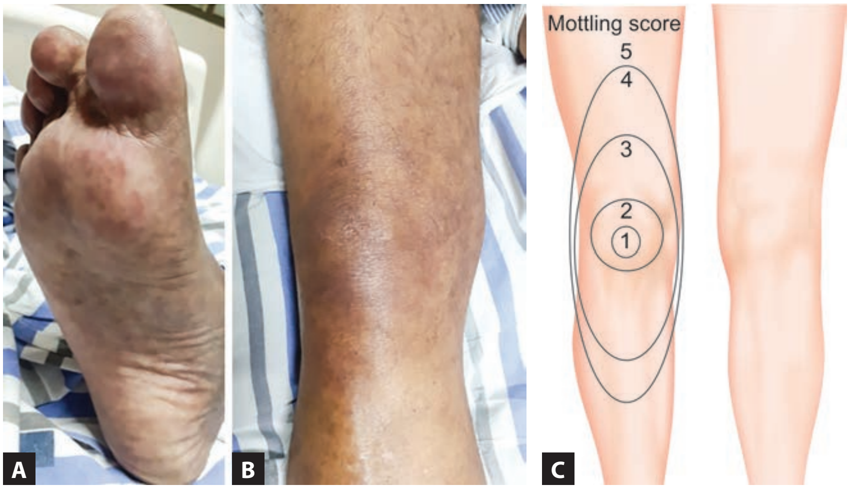


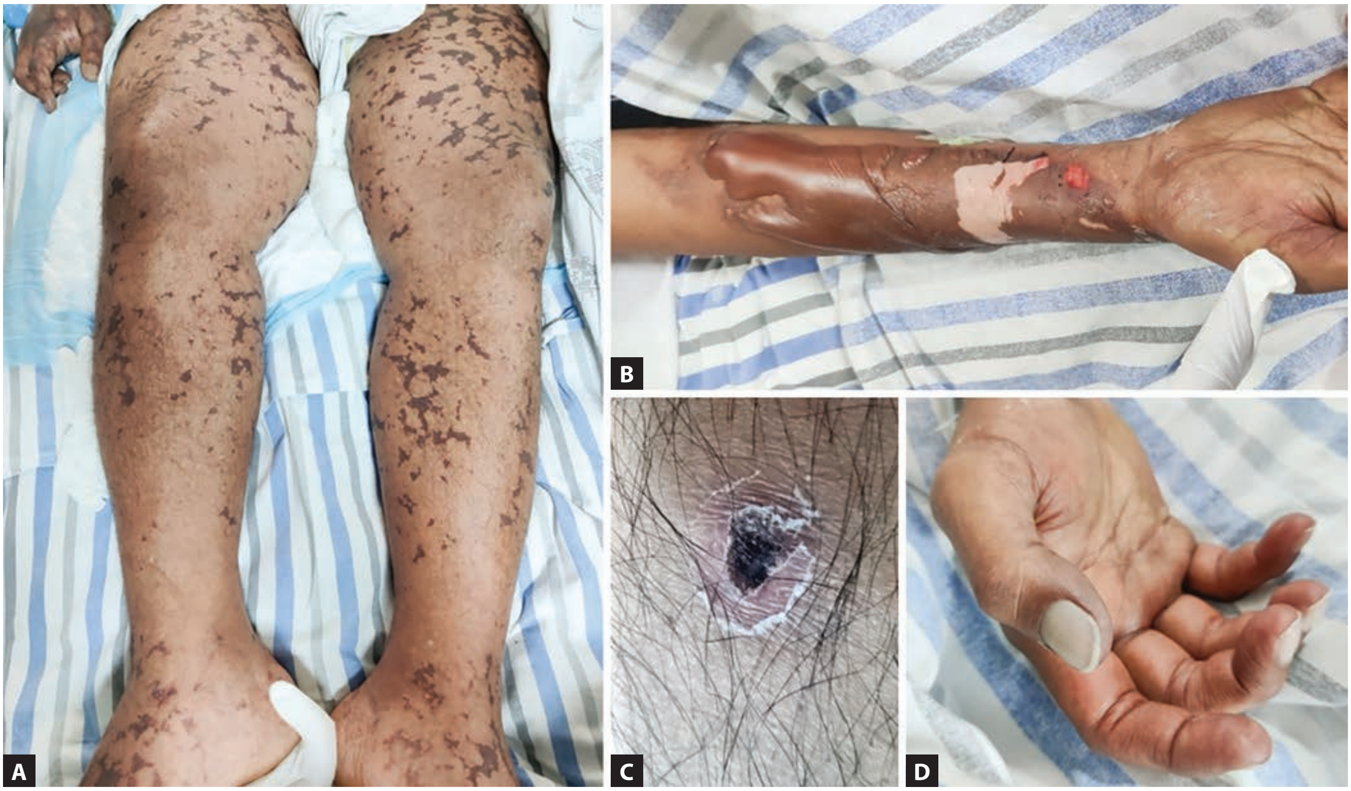
Fig. 2: Edematous limbs, pressure injuries—heels (discoloration, grade 1 pressure sore), scale skin. Features of long hospital admission. Discoloration of toes—severe shock and vascular compromise.



Skin mottling score

Score	*Skin mottling*
0	No mottling
1	Mottling about size of a coin over the center of the knee
2	Mottling over the knee, not extending above the upper margin
3	Mottling extending up, but below the mid thigh
4	Mottling up to the groin crease
5	Mottling extending above the groin

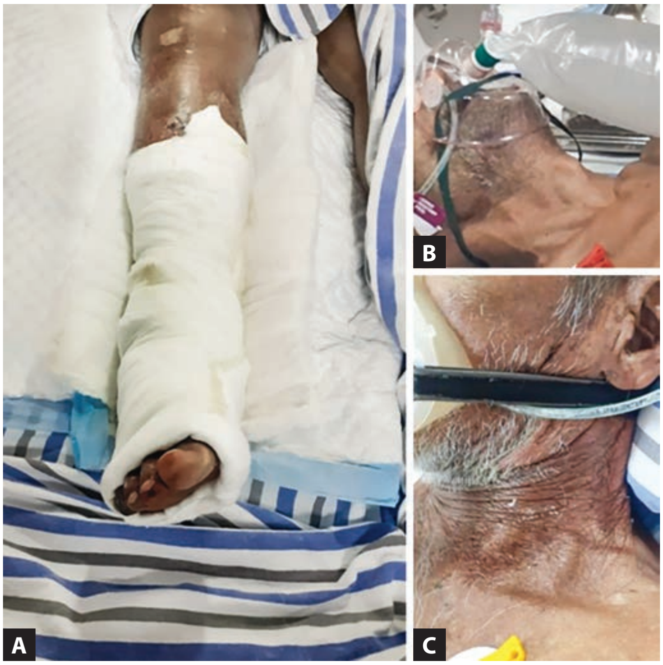
Figs. 3A to C: Discoloration of the skin due to shock. The right sole and knee. Mottling of skin over the knee and things are not easily appreciated in brown and dark-skinned individuals.



Figs. 4A to D: (A) Purpura fulminans; (B) Necrotizing fasciitis—bulla and necrotic patch; (C) Eschar—seen in scrub typhus; and (D) Cyanosis of digits.

Photograph suggestions:

- Accessory muscle use—sternocleidomastoid
- How to look for chest raise, ensure the airway is patent
- Dilated neck veins
- Gangrene limbs
- Necrotizing fasciitis



Figs. 5A to C: Postoperative necrotizing fasciitis. Accessory muscle use, active sternocleidomastoid, and dilated neck veins.

TABLE 3: Summary of general examination signs during rapid examination.

S. No.	*Domain*	*Description*
1.	Vitals	*Bedside monitors:* Any abnormal and alarming readings are usually the first thing the physician sees at the bedside and often picked up during continuous monitoring
2.	GCS	GCS is measured in all patients that gives a quick clue of brain function
3.	Head and neck	• *Flushed, dilated neck veins:* SVC syndrome/increased intrathoracic pressure • *Sweating, pale dusky face:* Increase in sympathetic activity as a compensatory response • *Pupils—unequal pupils:* Intracranial pathology • *Face swelling:* – Edema—renal or heart failure – Unilateral with crepitus—pneumothorax – Focal swelling below eyes with discoloration—? fungal sinusitis • *Malar rash:* SLE • *Edema of lips:* Angioedema • *Temporal wasting, no buccal fat:* Malnutrition, chronically ill patients • *Front of neck swelling:* Large thyroid compressing airway often have stridor. Auscultate the trachea for stridor and air movement • *Palpate carotid pulse and look for bruit:* Palpate for 5 to 10 s to differentiate between severe bradycardia and no pulse. Feeble pulse may indicate severe hypovolemia or poor cardiac output. Bruit in an unconscious patient may indicate cerebrovascular compromise
4.	Trunk and back	• Unequal chests raise rule out tension pneumothorax, a quick lung USG helps • *Breathing pattern:* – Apnea/bradypnea—respiratory compromise or impending cardiorespiratory failure – Irregular breathing—stroke – Deep breathing—acidotic patients • *Chest shape:* – Barrel-shaped—COPD

Contd...

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S. No.	Domain	Description
5.	Abdomen and genitals	• *Gross abdominal distension:* Gross ascites, centralization of volume, bowel pathologies • *Abnormal pulsations:* Abdominal aorta aneurysm • *Large ecchymosis at flanks:* Severe pancreatitis, abdominal wall cellulitis, and deep-seated infections
6.	Extremities	• *Signs of circulatory failure:* – Cold peripheries, increased CRT – Peripheral cyanosis – Mottling • *Discoloration and gangrene:* Infections, peripheral vascular disease, thromboembolic • Signs of cellulitis or necrotizing fasciitis • Pedal edema • *Fang marker or insect bite marks:* Envenomation
7.	Bed space or surrounding environments	• Knowledge of environment—especially helpful in cases of trauma, burns (closed space has risk of inhalational burns) • *Monitors and pumps:* – Temporary pacemaker, vasopressor infusions

(COPD: chronic obstructive pulmonary disease; CRT: capillary refill time; GCS: Glasgow Coma Scale; SLE: systemic lupus erythematosus; SVC: superior vena cava; USG: ultrasonography)

SUMMARY

1. Delay in recognizing clinical deterioration of a patient and delay in instituting appropriate therapy will be associated with poor outcome.
2. Scores like early warning scores can be used to trigger a review of medical personnel and escalate care of patients.
3. Familiarity in recognition of common life-threatening conditions and institution of appropriate therapy is the key to success.
4. Training and periodic update of the whole team including the nursing and other paramedical team members is important.

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Chapter

5

Communication in Intensive Care: The Soft Skills to Learn

Shivakumar Sankarnarayanan Iyer, Jignesh Navinchandra Shah

ABSTRACT

Intensivists require good communication skills from the time of admission to daily counseling to family meetings dealing with difficult situations and conflict and finally to help patients and families deal with death and dying. The principles of communication outlined in the Institute of Medicine (America) and the Kalamazoo statement include establishing a rapport with patient and family, giving information in easy-to-understand language, and recognizing and dealing with verbal and nonverbal cues that indicate distress and assist ethical and appropriate decision-making throughout the course of critical illness. Several tools and models like the "Setting-Perception-Invitation-Knowledge-Emotions-Summarizing" (SPIKES), the Cardiff six-point toolkit, and the "Naming-Understanding-Respect-Supporting-Exploring" (NURSE) model help us have a structured approach to communication in an intensive care unit (ICU).

INTRODUCTION

Evidence-based medicine is not only the use of the latest best evidence to guide therapy but also using good communication skills to elucidate the clinical circumstances and preferences of the individual patient and family.

Family members have rated communication as one of their most important concerns during critical illness. They rely on honest and accurate information about the disease process and its likely course for decision-making in an intensive care unit (ICU). Healthcare providers, however, fall short of the expectations of patients and families. Observational studies have shown that up to one-third of family members may not be satisfied with communication in the ICU. The factors that have been implicated include inadequate time, lack of consistent information, and conflicting information given by healthcare providers. Inadequate communication affects family satisfaction, impairs clinical decision-making, and worsens psychological outcomes of family members. The dissatisfaction of families may manifest as anger, violence, and litigation, which result in greater stress and burnout in healthcare providers. Many strategies have been suggested in order to improve family satisfaction in ICU.

In the assessment of a seriously ill patient, quick examination takes precedence over taking a history and establishing contact with the patient and family. Once the patient is resuscitated, connecting with the patient on a personal level and if that is not possible, explaining the gravity of the situation to the family and connecting with them is most important. This early connection aids the therapeutic process and the thread of communication guides the family through the ups and downs that patients and families experience during critical illness.

Understanding the principles of communication and applying them in the critical care setting is not easy and requires sustained effort and appropriate training.

CASE SCENARIO

A 65-year-old lady comes at 11.30 PM to the emergency department in a gasping state. She is resuscitated and simultaneously intubated and ventilated. A history of present illness and past comorbidities is obtained from her 70-year-old husband who reveals that she had urethral dilatation about a month back for stricture. She has a history of diabetes, hypertension, and ischemic heart disease. After resuscitation, sending cultures and starting appropriate antibiotics, she is shifted to ICU (Initial Communication).

The next day, she is on multiple vasopressors, requires mechanical ventilation with 80% FiO_2, and has a low urine output. The consultant counsels the husband and learns that their daughter will be arriving from abroad in a day or two. (Daily counseling) Over the next few days the patient's condition deteriorates and she now needs renal replacement therapy as well. The ICU consultant discusses the case with his team and other involved consultants and plans to discuss the treatment options with the husband and the daughter (Family meeting).

Situations in intensive care need communication skills. *Good communication is needed right through the course of acute illness*. Intensivists need to deal not only with patients/families but also with professional colleagues, junior and senior intensivists, nurses, respiratory therapists, administrators, counselors, and security guards among others. The principles outlined below deal primarily with doctor–patient/family interactions but may fruitfully be applied to other interactions as well. All they need is extra tact, understanding, and valuing each person as an important member in the healthcare team.

PRINCIPLES OF COMMUNICATION

The word communication comes to us from Latin, "communicate," which means to share or to receive. Communication has been described as "a process by which information is exchanged between individuals through a common system of symbols, signs, or behavior." A shared verbal language and an understanding of the components of nonverbal behavior is essential for good communication. Both these components are culturally and geographically determined and vary from place to place. Nonetheless, some common principles of good communication can be derived.

The four principles elucidated by the Institute of Medicine Committee on Quality of Health Care in America are described in **Box 1**.

The Kalamazoo consensus statement describes seven tasks that need to be done during the medical encounter **(Box 2)**.

Both these statements emphasize the importance of establishing an early relationship with the patient and family followed by exploring their understanding of the situation. It is vital to place this in the context of their preferences and values. The next step is providing honest and accurate information and arriving at a shared understanding of the problems and the care plan. Respecting patient and family autonomy and empowering them through shared decision-making is most important. All this helps replace the binary outcome of survival or death with family satisfaction.

BOX 1: Four principles—Institute of Medicine in America.

1. Obtaining and grasping the patient's viewpoint
2. Taking into account the patient's psychological state and social reality
3. Arriving at a joint understanding of the patient's clinical problem and looking at solutions in the context of the patients preferences and values
4. Respecting patient's autonomy and empowering the patient through shared decision making

BOX 2: The Kalamazoo consensus statement.

- Building the doctor–patient relationship
- Opening the discussion
- Gathering information
- Understanding the patient's perspective
- Sharing information
- Reaching an agreement on problems and plans
- Providing closure

MODELS AND TOOLS FOR GOOD COMMUNICATION

Setting, Perception, Invitation, Knowledge, Emotion, and Summary Model

The SPIKES (Setting, Perception, Invitation, Knowledge, Emotion, Summary) model was first elucidated by Baile et al. in 2000 for breaking bad news to cancer patients and their families. It has since been used extensively in several settings, including critical care **(Table 1)**.

Cardiff Six-point Toolkit

More recently, Pease et al. have set out the "Cardiff six-point toolkit" as a useful aid for ensuring good communication in end-of-life and palliative care communication **(Box 3)**.

Comfort

Making the patient/family comfortable before starting the interview is extremely important. It includes ensuring a quiet place, comfortable seating, a pleasant atmosphere, making water and tissues available, putting the phones off, and minimizing interruptions. Putting the patient/family at ease psychologically is also vital. During COVID-19 when

TABLE 1: SPIKES (Setting, Perception, Invitation, Knowledge, Emotion, Summary) model.

Principle	***Explanation***
Setting	Ensuring a comfortable physical and psychological setting
Perception	Exploring the patient/family understanding
Invitation	Judging the patient/family's readiness for receiving bad news
Knowledge	Delivering knowledge in a form that the patient/family can understand
Emotion	Watching/listening to verbal and nonverbal cues and acknowledging emotions
Summary	Summarize the meeting and draw up further plans

BOX 3: Cardiff six-point toolkit.

1. Comfort
2. Question style
3. Language
4. Listening/use of silence
5. Reflection/acknowledgment
6. Summarizing

communication was virtual, more effort was required to put the family at ease. An appropriate beginning would be to make introductions and ask a question like "How are things? or Shall we start?"

Question Style

One generally starts by asking an open-ended question that allows the patient or family to direct the content and pace of the conversation. It gives no idea of the expected answer. It also allows them to express their emotions. A typical example would be "How have things been?" If the patient or family chooses to answer such a question by enquiring about future difficult topics such as progression or prognosis, it helps us to take the discussion forward by asking further focused questions. On the other hand, if they respond emotionally, acknowledging the emotions and allowing their full expression would be appropriate.

Focused questions generally concentrate on a particular topic or symptom. One type of focused question is an exploratory question that describes a future possibility, for example, "Have you wondered what might happen if we are not able to manage your breathlessness with oxygen alone?"

Closed-ended or direct questions are utilized to clarify matters and usually have yes or no type responses.

Leading questions or asking too many questions simultaneously should be avoided.

Language

One must explore at the very beginning what language the family or patient thinks in. Language reflects the cultural, religious, and ethnic milieu of the family and helps us understand the patient or family's emotions and state of mind. Families often express themselves through their gestures, facial expressions, and tone of voice. In fact, spoken language may only be 7% of communication, the tone may be 33% and 60% may be nonverbal. Medical information should be conveyed in simple language and medical jargon should be avoided.

Listening/Use of Silence

Listening should occur 80% of the time and talking only 20% in ideal doctor–patient/family encounters. Active listening requires one to be calm, mindful, and to convey attention by making eye contact and using an open posture. Attentive silence allows families and patients to express their concerns and emotions. It allows them to process information and ask relevant questions. It may also encourage them to explore difficult or uncomfortable issues.

Reflection/Acknowledgment of Emotions

Reflection is a method by which the healthcare provider repeats a significant word or cue used by the patient/family and allows the patient or family to elaborate further on that topic. Sometimes reflection may facilitate the expression of the feeling behind that word. A question like "Am I going to die?," will need to be met by a question like "That is a really difficult question to ask, what makes you ask it now?" This acknowledges the distress without giving a simplistic answer. Appropriate silence after such an interaction may help the patient/family voice their concerns with greater ease.

Summarizing/Recapping

Information given in small chunks is preferred. Summarizing and recapping as we go along helps check understanding. Summarizing, offering support, and availability is a good way to end a meeting.

FIVE FUNDAMENTAL PRINCIPLES IN COMMUNICATION SKILLS

Robert Arnold et al. have delineated five important principles in their critical care communication (C3) course that are similar to the Cardiff toolkit and the SPIKES model **(Box 4)**.

"Ask-Tell-Ask"

The healthcare provider *asks* the patient/family to describe their comprehension of the problem by asking open-ended questions. The patient/family is thus given a chance to voice their concerns and their understanding is explored. Asking for their permission also helps to establish mutual trust. This helps negotiate a common agenda for the meeting and elucidates the informational and decision-making style of the family. Further, we *tell* the patient/family in easily understood language about the problem. It is important to avoid the use of medical jargon. Finally, we *ask* what they have understood and provide further clarification if required.

"Tell Me More"

Families and patients are trying to comprehend serious issues at several levels. At a superficial level, the family is processing the information. At a deeper level, the information evokes emotional responses. At the deepest level, the inner discussion may be about what the new information means to the patient or family member in terms of their own self. "Tell Me More" is a strategy that allows us to explore these levels—What more information do you need? How does this make you feel? How will this affect you in your day-to-day life?

"Using Reflections"

Rephrasing a patient/family statement can be a powerful technique for raising further concerns or exploring understanding. Simple reflections may just repeat what the family member said:

Mother: I can see what you mean but how can I give up hope?

Doctor: Give up hope?—(Reflection)

Mother: What will I do if he gets sicker or does not make it?

Complex reflections explore what the family member is saying and may incorporate the clinicians' perspective. This may bias the family member, but may also help change the perception of the family member.

Patient's brother-in-law: I get what you are saying doctor, but please—don't tell my sister, she will be devastated and won't be able to handle it.

Doctor: I can see that you care deeply for your sister and want her to be prepared to accept the situation. (Empathic and kind manner and allowing enough silence)

Patient's brother-in-law (breaks into tears): She has two small children doctor, I don't know how she will cope.

Thus, reflections can express sympathy and understanding and may allow the patient/family to direct the discussion and express their views.

"Responding to Emotion"

Discussions about end-of-life are often challenging because of the intense emotional upheavals that occur when faced with imminent or actual loss. As clinicians, not only do we have to navigate anger, sadness, grief, disbelief, hopelessness, and despair of families but also be mindful of our own feelings around death and dying.

This allows us to actively listen to patients/families and jointly experience their angst. We are able to direct our attention to explore and handle patient/family needs.

Patients/families often express emotions through nonverbal gestures and indirect verbal cues. Identifying and responding to these is extremely vital. Emotional outbursts interfere with the ability to process information and may profoundly impair decision-making ability. Once such an emotional reaction is identified, it must be addressed as soon as possible.

One of the described models for this is NURSE (Naming, Understanding, Respecting, Supporting, and Exploring) **(Table 2)**.

The most effective empathic statements are those that link the "I" of the healthcare provider to the "you" of the patient/family. We need to be able to put ourselves in the position of the patient/family and understand them fully.

BOX 4: Five principles.

1. Ask-tell-ask
2. Tell me more
3. Using reflections
4. Responding to emotion
5. Assessing the informational, coping, decision-making style

TABLE 2: NURSE (Naming, Understanding, Respecting, Supporting, and Exploring) model for acknowledging emotions.

Strategy	*Explanation*
Naming	Name the emotion appropriately, e.g., *"Some people in this situation would be upset"* rather than *"I can see that you are angry."* (People don't like to be told what they are feeling)
Understanding	Understand and appreciate what the patient/family is experiencing, e.g., *"I cannot begin to imagine what it might be like' to be in this situation."* Avoid giving false hope or premature reassurance
Respect	Showing respect can be a nonverbal response but a verbal response can help validate the emotion. Depending on the strength of the expressed emotion you might want to give an equally strong acknowledgment. Praising the way that the patient/family is coping is a good way of showing respect
Supporting	Support can be expressed in many ways. Willingness to help, being available, expressing your concern, and making statements about working together for resolution are all very useful. For example, *We have covered a lot of ground today. If you want to ask me anything else I will be available in my office and of course we have a scheduled meeting for tomorrow*
Exploring	Exploring what people are feeling with open-ended questions or inviting them to share their story is a good way to help them express their emotions. For example, *"What has this been like for you?" "I have given you a lot of information. What are you thinking?"*

For example, "I" can understand how difficult this must be for "You."

"Assessing the Informational, Coping, Decision-making Style"

Patients and families process information and cope with crisis situations in vastly different ways. Two distinct types of informational coping style "Monitors" and "Blunters" were identified by Miller et al. by using the Miller Behavioural Style Scale. "Monitors" are family decision makers who seek huge amounts of information and are likely be more anxious or depressed. They will require help with identification of their emotions and support for prevention of depression. On the other hand, "Blunters" avoid too much information, place themselves as far away from the situation at hand as possible, and are likely to be in denial. They are usually not able to take decisions. Appropriate decision-making in "Blunters" is driven by helping them to deal with their emotions and then understand information. At times, however, decision-makers may exhibit a combination of styles. It is important to explore this:

"Some people prefer having all the information that they can get whereas others get overwhelmed with too much information and are content getting to know about the big picture. What kind of person are you?"

Often we may have to try out one or the other approach and decide which approach is best at that particular time for that particular person.

APPROACH TO COMMUNICATING WITH CRITICALLY ILL PATIENTS AND THEIR FAMILIES

History Taking

Critically ill patients come to the ICU either directly from the emergency department, from the wards due to acute deterioration, or from the operation theater. They may have a background illness or may have fallen ill for the first time. The first step is usually resuscitating a patient and is followed by a more detailed history and examination. History taking in the ICU is often a challenge. It is not only crucial in deciphering the clinical circumstances of critical illness, but also in understanding its meaning for the patient and family. **Box 5** summarizes the points that need to be gleaned from the history in ICU patients.

Daily Counseling

Families must be spoken to daily at least once in a formal sit-down session by the faculty in charge of that patient. The family member with whom this communication happens should be designated beforehand and should ideally be present at all these daily counseling sessions for continuity. **Box 6** suggests a format for a daily counseling session.

Family Meeting—A Tool to Address Difficult Issues and Conflict

Family meetings should be conducted within 24–48 hours of an ICU admission. The other situations that necessitate

BOX 5: History taking in intensive care unit (ICU) patients.

- Is the illness acute or acute on chronic?
- If there is a chronic illness, is it now life-limiting?
- Has the illness started with a single organ and become systemic or was it systemic to begin with?
- What are the comorbidities or addictions?
- What is the educational, socioeconomic status of the patient and family?
- Is there any form of health insurance that the patient has?
- What is the socioeconomic impact of the patient's illness on the family?
- How has the illness affected the patient and the family psychologically and emotionally?
- Who are the various medical professions involved in the care of the patient?
- Who are the different family stakeholders in the care of the patient?
- Is there a trusted medical professional in the family who can help communication?

BOX 6: Daily counseling session.

- Faculty-in-charge of patient should speak to a designated family member at least once a day
- It should be a sit-down session preferably at a designated time in a comfortable setting
- Input from other consultants, junior doctors, nurses, social worker, and administration should be collated prior to the meeting
- Prior understanding should be explored before giving information
- Information should be given in simple language without medical jargon in small chunks always checking for comprehension
- Emotions should be acknowledged and dealt with empathy
- The session should end with a brief summary and who will provide information and support in case of a change in status before the next counseling session

a family meeting include a significant change in the patient's status, disagreement among family members, and when a transition of goals of care to palliative treatment is thought necessary.

There are several steps in conducting a structured family meeting:

- *Identify and invite the clinicians/providers involved in the treatment:* Other members of the team such as nurses, counselors, or social workers should be included if necessary. An involved family doctor could provide invaluable help. Palliative medicine consultants can help in difficult situations, especially if there is conflict.
- *Identify the appropriate surrogates:* The patient should be included but this is frequently not possible in an ICU. In India, families are large and sometimes, especially in rural families, community members who are providing financial and social support may need to be considered.
- *Meet prior to meeting the family and decide who will lead the meeting:* Resolving and acknowledging conflict and uncertainty in the treatment plan within the team, and discussing the agenda for the family meeting are very important. Other professionals such as counselors can provide valuable information about the family.
- *Decide an appropriate meeting place*
- *Consider and appreciate the family dynamics*
- *Make introductions and explain the goals for the meeting*
- *Ask what the family already knows*
- *Build on what they already know* by providing further information. Provide information in small chunks.
- *Check their understanding.*
- *Summarize the current situation:* Provide detailed information regarding the current diagnosis, the treatment being given and the prognosis. Discussing the prognosis is always challenging. Asking families how much information they would like can be a good beginning. Families are usually more hopeful than doctors in their estimates. It is best be open-minded as there is a lot of uncertainty in the ICU setting. We must try to come to a consensus that reflects patient and family interest. In the ICU, a time limited trial of aggressive therapy can resolve uncertainty regarding the course. Another way of dealing with disagreement is to use "Hope for the Best, Prepare for the Worst" type of statements. The care plan should be prepared by marrying the clinicians' medical and technical expertise and the patient and family's values and goals.
- *Consider end-of-life issues empathically and in a joint manner:* It is paramount to help families understand that they should be guided by what the patient would have wanted in a particular situation. A shared decision based on medical facts and patient values helps move the decision-making burden from the family and reduces their guilt.
- *Manage emotions:* The NURSE mnemonic has already been discussed as an aid for managing emotions. Another useful mnemonic is VALUE that portrays a communication method that improves harmony between care providers and surrogates/family.

The elements include valuing surrogate/family communication (V), acknowledging their emotions with reflective summary statements (A), listening actively (L), understanding the patient as a person with the help of open-ended questions (U), and eliciting questions (E).

- *Identify and resolve conflict:* Emotions such as anger, frustration, stress, denial, grief, guilt, lack of trust, and misunderstanding often accompany conflict between healthcare providers and surrogates. Addressing these emotions with empathy and providing more time often helps to resolve conflict. On occasion, another opinion, an ethics committee referral, or palliative medicine consultation may be needed.
- *Summary and further plan:* Before concluding the meeting, it is important to note down the decisions made, the areas of conflict, if any, and the plan for follow-up.
- *Conducting a debrief with the team:* This is a good method to teach and learn regarding communication. Ethical issues should also be noted and any moral angst experienced by the team members is shared.

Dealing with Dying and Death and the Process of Grief

Elisabeth Kubler Ross has described five stages of grief in her seminal work on death and dying, namely, denial, anger, bargaining, depression, and acceptance. These stages of grief are now thought to be ways of coping with loss and need to be identified so that they may be tackled appropriately. In the ICU, death is often unexpected, premature, and traumatic and is a risk factor for pronged and sometimes uncomplicated grief. Providing empathic psychologic and emotional support for the family is important and needs application of the principles of good communication. Occasionally, specialized psychologic support techniques such as cognitive behavioral therapy or medications may be needed to manage prolonged or complicated grief.

SUMMARY

Several tools and guidelines are available for intensivists in order to facilitate good communication. Soft skills for communication however need to be taught in a simulated environment in workshops that use standardized patients. Practice under supervision will then help hone these skills and help provide optimum care for ICU patients.

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Chapter

6

An Algorithmic Approach to Symptomatology in Critically Ill Patients

Arvind Rajamani

ABSTRACT

Diagnostic decision-making in acute care medicine typically requires physicians to make rapid diagnoses to provide supportive and specific care for patients having life-threatening conditions. As the symptoms of acute health conditions may often be nonspecific and shared across organ systems, it is important to start with broad differential diagnoses. At the same time, unless the physician is able to rapidly identify the patient's specific diagnosis, there may be delays in providing specific therapies. Hence, it is important to employ a structured approach to diagnostic decision-making.

INTRODUCTION

In this chapter, the author presents an approach that starts broad and ends narrow—like an inverted pyramid. The components required for this process are as follows:

- Identifying the potential organ systems affected by associating the patient's presenting symptoms with the typical symptoms associated with the various organ systems in the body
- Making syndromic diagnoses for each organ system
- Making a *categorized* list of pathological conditions/diseases associated with that syndrome; these diseases are categorized either into etiopathogenetic classes or anatomic classes (depending on the clinical syndrome that is being considered)
- Having a pattern analysis of the common ways by which these diseases present in terms of their specific history, physical examination findings, and clinical course.

At every stage, remember these golden rules of medicine:

- *At every stage, start broad, then go narrow*. If you do not have a broad range of differentials to start with, you are more likely to make errors. On the other hand, if you do not have the confidence to narrow down to the smallest number of possible differentials, you will end up becoming confused due to too many possible differentials.
- *Your diagnosis does not have to be correct all the time*. Do not ask yourself if the diagnoses are correct or incorrect. Instead, ask if the diagnoses are *likely or unlikely* in the patient's clinical context. For instance, if you hear hoof beats in a horse racing event, they are likely horses rather than zebras. However, if you hear hoof beats in the savannahs of Africa, it is more likely that they are zebras rather than horses. *Therefore, context is everything!* In other words, anything that is not logical must be reconsidered.
- *Uncommon manifestations of common diseases are still more common than common manifestations of uncommon diseases!* Think this statement over in your head. We will come back to this in each of the case scenarios at the end.

Before constructing our pyramid, let us first understand these steps in more detail.

CASE SCENARIOS

	Presenting symptoms and initial clinical findings	*Provisional diagnosis by junior doctor*
Case scenario 1	A 65-year-old man presents with light-headedness, nausea, and vomiting that started 2 hours ago *Vital signs in triage:* • Drowsy and mildly confused • BP 82/44 mm Hg • Heart rate 38 beats/min • O_2 saturation on room air = 100%	Could not make a diagnosis
Case scenario 2	A 65-year-old woman with a history of type 2 diabetes mellitus presents with oliguric acute kidney injury. She has been generally unwell with no other specific symptoms for approximately 10 days *Vital signs in triage:* • Deeply comatose • BP 139/73 mm Hg • Heart rate 76 beats/min (sinus) • O_2 saturation on room air = 100% • pH 6.9 • $paCO_2$ 54 mm Hg • Base excess = –19 mmol/L • Lactate = 17 mmol/L	Septic shock with multiorgan failure causing AKI and lactic acidosis
Case scenario 3	A 33-year-old woman with no previous comorbid conditions presents with a 1 week history of generalized unwellness. She reports easy bruisability over the past 48 hours *Vital signs in triage:* • Fully conscious and oriented • BP 120/78 mm Hg • Heart rate 146 beats/min (sinus) • Temperature = 39°C (102.2°F) • Respiratory rate = 28 breaths/min • O_2 saturation on room air = 100%	Septic shock with multiorgan failure

(AKI: acute kidney injury; BP: blood pressure)

Basic Knowledge of Symptoms Associated with each Organ System

The human body has six organ systems relevant to acute care medicine. **Table 1** outlines the key organ systems with the organs that populate them and the associated clinical syndromes for the organ systems. **Box 1** gives a list of common pathologies associated with these clinical syndromes and the specific symptoms on history, physical examination signs, and investigations typically associated with these syndromes.

CASE SCENARIO 1

A 65-year-old man presents with light-headedness, nausea, and vomiting that started 2 hours ago.

Vital signs in triage: He is drowsy and mildly confused; BP 82/44 mm Hg; heart rate 38 beats/min. O_2 saturation on room air = 100%.

History and Symptomology

As with any scenario in medicine, it is imperative to begin with a history and examination. Depending on the acuteness of the situation, the history needs to be targeted and specific initially and can be explored in further detail, if needed, once the patient is stable. It is not always possible to take a history from the patient directly, such as in our patient above who is found to be confused and mildly confused at the scene. Collateral resources such as family members, ambulance notes, witness accounts, and local doctor can be utilized. A framework is provided

TABLE 1: Key organ systems chart with associated clinical syndromes for the organ systems.

Organ system	***Organs (in order of common presentations)***	***Typical clinical syndromes***	***Categorized list of pathologies associated with these syndromes***	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
Nervous system	*Central nervous system, comprising:* • Brain • Meninges	• Altered level of consciousness—spectrum ranging from confusion, drowsiness, to coma • Motor or sensory deficits	Structural lesions in CNS that affect consciousness—bilateral reticular activating system/brain stem	Focal neurological deficits	Sudden onset	With or without focal neuro deficit	• Brain imaging—MRI, CT perfusion scan • EEG
			• Nonstructural CNS pathologies • Non-neurological pathologies that affect consciousness	No focal neurological deficits	• Variable onset • Symptoms associated with other organ systems	• No specific neurofindings • Findings associated with other organ systems	Depends on likely organ system
	Peripheral nervous system, comprising: • Spinal cord • Nerve roots • Peripheral nerves • Neuromuscular junction • Muscles	Motor or sensory deficits with preserved higher functions	Spinal cord syndromes	Focal neurological deficits associated with spinal cord level	• Typically, acute onset • May have prodrome of back pain	Cord dermatome and myotome level	MRI spinal cord
			Nerve roots and peripheral nerve disease have specific patterns of motor and/or sensory deficits	Other diseases have their typical patterns	• Variable onset • For example, GBS presents as bilaterally symmetrical flaccid paralysis while myasthenia has other prodrome symptoms	Lower motor neuron signs	• Nerve and muscle electrophysiological studies (demyelination/denervation/myopathic pattern) • CSF

Cardiovascular system	• Most common—left and right ventricle
	• *Less common:*
	  – Pericardial sac
	  – Valves
	  – Aorta
	  – SVC and IVC
		Shock (forward failure)	Hypovolemic shock	*Must* be associated with specific hypovolemic reason	↓ intake/↑ losses/significant fluid shifts	• Signs of shock (hemodynamic instability, confusion, oliguria with concentrate urine), *plus*
						• Signs of dehydration (cool peripheries, poor skin turgor, and low JVP)
							• ↑ lactate is a part of any cause of shock
							• Specific to hypovolemia
							  – Low CVP
							  – Pulse pressure variation on arterial line or plethysmograph
							  – Small, collapsing IVC on echo

			Hemorrhagic shock	*Important to identify bleeding source*	History suggestive of source of bleeding	As above, plus signs of hemorrhage	• ↓ Hb (*Note*—Hb concentration may be normal initially due to whole blood loss) • Later stages—consumption coagulopathy/DIC • eFAST

Contd...

Contd...

Organ system	***Organs (in order of common presentations)***	***Typical clinical syndromes***	***Categorized list of pathologies associated with these syndromes***	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
			Obstructive shock	DDx—tension pneumothorax, pericardial tamponade	• *Sudden* onset • Pneumothorax → hypoxemia plus hypotension • Tamponade →	• Pneumothorax signs (↓ chest expansion, tympanic percussion note, ↓ breath sounds, raised JVP) • Tamponade → raised JVP, muffled heart sounds	• Pulse pressure variation on arterial or plethysmograph trace • Ultrasound confirmatory
			Cardiogenic shock	ACS	Acute onset anginal chest pain	Nothing specific	• ECG—ST/T changes • Echo—SWMA
				Less common —LVOTO	• Increasing catecholamine requirement • May have HCM	Nothing specific	Echo
				Uncommon—severe valvular stenosis/ acute regurgitation, typically, mitral and aortic valve	• Stenosis—chronic history • Acute regurgitation—history for cause (valve rupture and chordal rupture)	• Typically, left heart congestion • Note—typical valve stenosis signs may be absent in low cardiac output states	*Echo:* • Stenosis—thickened or deranged valve morphology plus severely reduced opening • Acute severe regurgitation—usually has associated morphological abnormality (flail leaflet, ruptured chordal tendon, vegetation)
				Right heart—ACS, submassive/massive PE	• Acute onset dyspnea and hypoxemia • Hemodynamic instability (variable finding) • *Common risk factors:* – Immobilization – Critical illness – Cancer – Medications	• DVT signs • Peripheral edema (however, this may not be present in acute state)	• ECG signs of PE (note—may not be present) • *Echo:* RV dilation, ↓ RV function • Lower limb venous ultrasound for DVT

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Organ system	Organs (in order of common presentations)	Typical clinical syndromes	Categorized list of pathologies associated with these syndromes	Specific diagnostic criteria			
				Pattern	**Specific history**	**Specific examination**	**Specific investigation**
			Distributive shock: • Vasodilatory – Septic – Anaphylactic – Spinal • Vasoconstrictive – Neurogenic	Septic	• May present as "warm" shock or "cold" shock—this is *not* a distinguishing feature • Features of infection • Source of infection	Focus of infection	• Two sets of blood cultures • Relevant body fluid cultures • Nonculture microbiological investigations • Relevant imaging
				Anaphylactic	• Trigger for anaphylaxis • Features of allergy	Features of allergy	↑ mast cell tryptase
				Spinal (uncommon)	Acute spinal cord trauma/cord compression	Lower motor neuron type of paralysis	MRI spine
				Vasoconstrictive (rare)	Intracranial emergency	• Hypertension • Cold peripheries	Brain imaging
		Congestion (backward failure)	Left heart congestion	APO due to acute CCF or decompensation of chronic CCF	• Acute onset of dyspnea and hypoxemia • Hemodynamics may be normal or may be unstable	Signs of pulmonary edema	• *CXR signs of pulmonary edema:* – Interstitial edema – Kerly B lines – Pleural effusion if chronic • *Lung ultrasound:* – Transudative B-line pattern – Pleural effusion if chronic
			Right heart congestion	Peripheral edema	• Acute onset of dyspnea • Hypoxemia not essential, but may be a part of chronic illness • Hemodynamics may be normal or may be unstable	Signs of right heart failure	*Echo:* • RV/RA dilation • RV hypertrophy if chronic • RV function is variable • Dilated, noncollapsing IVC • TR and PHTN

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Organ system	Organs (in order of common presentations)	Typical clinical syndromes	Categorized list of pathologies associated with these syndromes	Specific diagnostic criteria			
				Pattern	Specific history	Specific examination	Specific investigation
		Electrical dysfunction (dysrhythmias)	Broad QRS complex	Monomorphic	• *Sudden onset of any of these:* – Palpitations – Hemodynamic instability – Syncope – APO • *Risk factors:* – IHD – DCM – HCM – Chaga disease	No specific clinical findings other than of the cause	• ECG • Biochemistry
				Polymorphic	• Sudden onset of same symptoms as above • *Risk factors:* – Long QTc – IHD	As above	As above
			Narrow QRS complex tachycardia	*Regular:* • Sinus • Atrial • Atrial flutter • AV node re-entry • AV re-entry • Junctional	• Sudden onset of same symptoms as above • *Risk factors:* – Infection – Hyper-thyroidism – Lung disease – Medications (e.g., digoxin, theophylline) – Hypoelec-trolytemia (Mg, K)	As above	As above
				Irregular: • Atrial fibrillation • Multifocal atrial tachycardia	• Sudden onset of same symptoms as above • Similar risk factors	As above	As above

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Organ system	**Organs (in order of common presentations)**	**Typical clinical syndromes**	**Categorized list of pathologies associated with these syndromes**	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
Respiratory system	• Lungs • *Less common:* – Pleura – Chest wall components – Upper airway	Hypoxemic respiratory failure	Intrapulmonary shunt	*Alveolar consolidation:* • Most common—infective (pneumonia) • *Noninfective consolidation:* – Pulmonary hemorrhage – Autoimmune – Pulmonary contusion	• *Infective pneumonia:* – Fever – Cough and/or sputum and/or dyspnea • *Pulmonary hemorrhage and autoimmune:* – Fever – Bloody sputum – Systemic involvement • *Pulmonary contusion:* – Chest trauma	• ↓ chest expansion • Dull note percussion • Bronchial breathing • ↑ vocal resonance • ↓ lung compliance on ventilator	• Cultures from blood, sputum, and pleural effusion, if present • Urinary antigens for *Pneumococcus, Legionella* • NAT/PCR for respiratory viruses • *Chest imaging (CXR/CT/lung ultrasound):* – Alveolar pattern of infiltrate – Air bronchograms – No loss of lung volume/deviation of mediastinal structures
				Alveolar atelectasis	*Risk factors:* • Thoracic surgery/trauma • Pain • Obesity • Muscle weakness • Increased sputum production • Endobronchial occlusion from sputum plug/foreign body • *Extrinsic compression:* – Pleural effusion – Abdominal distension – Symptoms—dyspnea, hypoxemia	• ↓ chest expansion • Deviation of trachea ipsilateral (inconsistent finding) • Dull note percussion • Bronchial breathing • ↑ vocal resonance • ↓ lung compliance on ventilator	*CXR/CT:* • Loss of lung volume ipsilaterally • Air bronchograms • Ipsilateral deviation of mediastinal structures (trachea, heart, and diaphragm)
				Pulmonary embolism	See above	Nothing specific	See above

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Organ system	***Organs (in order of common presentations)***	***Typical clinical syndromes***	***Categorized list of pathologies associated with these syndromes***	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
			Rare—extrapulmonary shunt	Intracardiac right to left shunt	Chronic history of hypoxemia	Signs of chronic hypoxemia	• Echo with Doppler • Echo bubble test (agitated saline contrast) → rapid crossover of bubbles from right to left heart within three cardiac cycles
				Hepatopulmonary syndrome	Cirrhosis and chronic liver failure	• Platypnea • Orthodeoxia	Echo bubble test (agitated saline contrast) → delayed crossover of bubbles from right to left heart after five cardiac cycles
		Hypercarbic respiratory failure	Decreased total minute ventilation	• Neuromuscular/musculoskeletal causes – *CNS:* - Reduced level of consciousness - Brainstem lesions – Nerve root, neuromuscular junction – Muscle • *Chest wall:* – Obesity – Pain – Pleural effusion – Abdominal distension	See above for each risk factor	See above for each risk factor	See above for each risk factor
			Decreased minute alveolar ventilation (i.e., increased dead space ventilation)	COPD/asthma	• Chronic history • Acute exacerbating condition (e.g., infection, heart failure, etc.) • Wheeze • Symptoms of acute exacerbating condition	• Expiratory outflow obstruction • Wheeze	• Nothing specific • CXR may show variable signs consistent with COPD or asthma

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Organ system	***Organs (in order of common presentations)***	***Typical clinical syndromes***	***Categorized list of pathologies associated with these syndromes***	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
Abdomen	• Gastrointestinal system (i.e., gut) • Hepatobiliary and exocrine pancreas	Acute abdomen due to perforated viscus	Perforation of any part of gastrointestinal tract	Peritonism	• Acute onset abdominal pain • Vomiting, constipation, ileus (variable)	Peritonism—guarding, rigidity, tenderness, and rebound tenderness	• CXR/CT abdomen for air under diaphragm • CT abdomen with oral and IV contrast may sometimes show location of air leak
		Other inflammatory causes apart	• Bowel obstruction • Ischemic gut • Biliary sepsis (acute cholecystitis, cholangitis) • Acute pancreatitis • Acute hepatitis	Nonperitonitic acute abdomen pattern	• Acute to subacute onset abdominal pain • Vomiting, constipation, ileus (variable)	• Nonspecific mild tenderness • Guarding, rigidity typically absent • Rebound tenderness absent	CT abdomen with oral and IV contrast
Genitourinary system	Renal tract	Acute kidney injury	• Prerenal • Postrenal • Renal	• Oliguria • Less common, nonoliguric AKI	• Acute to subacute onset • Cause/risk factors – Dehydration – Shock – Nephrotoxins – Chronic renal/ systemic diseases – Cell lysis – Others	• Nothing specific • Look for findings related to the cause	• Serum urea, creatinine, K+, ABG for HAGMA • Investigations for the cause
		Urinary tract infections	• Cystitis • Acute pyelonephritis • Prostatitis	Nothing specific	• Fever • Burning sensation while passing urine	• Nothing specific • Upper renal tract may be tender	• Blood and urine cultures • Renal tract ultrasound/CTKUB
	Genital tract		• *Males:* – Epididymo-orchitis – Prostatitis – Testicular torsion • *Females:* – Pelvic inflammatory disease – Ectopic pregnancy – Puerperal sepsis	Nothing specific	Torsion → acute/ sudden onset lower abdominal pain and vomiting	• Non-peritonitic pattern of acute abdomen • Prostatitis → tenderness on prostate per rectal examination	• Blood and urine cultures • Renal tract ultrasound/CTKUB

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				Specific diagnostic criteria			
Organ system	***Organs (in order of common presentations)***	***Typical clinical syndromes***	***Categorized list of pathologies associated with these syndromes***	***Pattern***	***Specific history***	***Specific examination***	***Specific investigation***
Hematological system	• Red blood cells (RBCs) • White blood cells (WBCs) • Platelets	• Anemia • Thrombocytopenia • Leucopenia	Bleeding	Nothing specific	• History of trauma/ surgery • Spontaneous bleeding • Often has pain in site of bleeding	Nothing specific	See hemorrhagic shock above
			Consumption (e.g., increased clotting)	Nothing specific	As above	Nothing specific	• ↓ Hb and platelets • Coagulopathy
			Increased destruction: • Hemolysis • Splenomegaly	Nothing specific	Nothing specific	Nothing specific	*Hemolytic screen:* • Schistocytes on blood film • ↑ bilirubin • ↑ LDH • ↑ K
			Decreased production of hematological cells: • Bone marrow disorders • Nutritional deficiencies	Chronic	Nothing specific	Subacute history	• Bone marrow biopsy • Blood levels
Metabolic and endocrine systems, mainly hypothalamo-pituitary, thyroid, adrenal, and endocrine pancreas	• Hypothalamo-pituitary • Thyroid • Adrenal • Parathyroid • Endocrine pancreas	• Hypo- and hyperpituitarism • Hypo- and hyperthyroidism • Hypo- and hyperadrenal • Hypo- and hyper-parathyroidism • Diabetic emergencies	Hypopituitarism, hypothyroidism and hypoadrenalism manifest as hypometabolic state	• Hypopituitarism may be acute (postshock, peripartum, and posthypophysectomy) • Severe hypo-thyroidism and hypoadrenalism present as acute decompensation of chronic conditions	• ↓ LOC • Hypotension • Sinus bradycardia • Hypothermia • Hypoglycemia • Additionally, hypopituitarism may have diabetes insipidus	• Hypothyroidism—possible goiter • Hypoadrenalism—possible skin pigmentation	• TFT - ↑↑↑TSH, ↓T4 • ↓↓ plasma cortisol • Hypopituitarism—specialized tests; brain MRI
			Hyperpituitarism and hyperthyroidism manifest as hypermetabolic state	Agitation, delirium	• Thyrotoxicosis → tachycardia, HTN, sweating, diarrhea • Hyperpituitarism—similar	Nothing specific	Thyrotoxicosis—↓↓ TSH

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Organ system	**Organs (in order of common presentations)**	**Typical clinical syndromes**	**Categorized list of pathologies associated with these syndromes**	***Specific diagnostic criteria***			
				Pattern	***Specific history***	***Specific examination***	***Specific investigation***
			Pheochromocytoma presents as sympathetic excess		Pheochromocytoma → episodic and/ or sustained severe hypertension	Nothing specific	Specialized tests
			Diabetic emergencies	DKA/HHS spectrum	• ↓ LOC • Kussmaul breathing • Symptoms of precipitant	• Dehydration • Findings of precipitant	• ↑↑↑ BSL • DKA → ↑↑↑ ketones and HAGMA • HHS → mildly ↑ ketones
				Hypoglycemia	• ↓ LOC • Seizures • Sympathetic stimulation symptoms	Nothing specific	↓↓↓ Blood sugar

(ABG: arterial blood gas; ACS: acute coronary syndrome; APO: acute pulmonary edema; AV: atrioventricular; BSL: blood sugar level; CCF: congestive cardiac failure; CNS: central nervous system; CSF: cerebrospinal fluid; CT: computed tomography; CVP: central venous pressure; CXR: chest X-ray; DCM: dilated cardiomyopathy; DDx: differential diagnosis; DKA: diabetic ketoacidosis; DVT: deep vein thrombosis; ECG: electrocardiogram; Echo: echocardiogram; EEG: electroencephalogram; eFAST: extended focused assessment with sonography in trauma; GBS: Guillain-Barré syndrome; HAGMA: high anion gap metabolic acidosis; Hb: hemoglobin; HCM: hypertrophic cardiomyopathy; HHS: hyperosmolar hyperglycemic state; HTN: hypertension; IHD: ischemic heart disease; IV: intravenous; IVC: inferior vena cava; JVP: jugular venous pressure; KUB: kidneys, ureters, and bladder; LDH: lactate dehydrogenase; LOC: level of consciousness; LVOTO: left ventricular outflow tract obstruction; MRI: magnetic resonance imaging ; NAT: nucleic acid amplification test; PCR: polymerase chain reaction; PE: pulmonary embolism; PHTN: pulmonary hypertension; RA: right atrial; RV: right ventricular; SVC: superior vena cava; SWMA: segmental wall motion abnormalities; TFT: thyroid function test; TR: tricuspid regurgitation; TSH: thyroid-stimulating hormone) below for history-taking and symptomology for cases such as scenario 1 **(Table 2)**.

Before speaking with the patient, use the information (symptoms and sign) you have and narrow down the body systems you would like to explore to guide your history taking. Getting to the bottom of our inverted pyramid, in other words, narrowing down the diagnosis is a dynamic process, meaning, that with every question, you should actively prioritize your differential diagnosis on a scale of most likely to least likely **(Table 3)**.

BOX 1: Etiological classification of diseases.

- Inflammatory, further subclassified as infective and noninfective
- Traumatic, including surgery
- Neoplastic
- Ischemic
- Allergic/anaphylactic
- Autoimmune
- Metabolic/endocrine
- Fluid and electrolytes
- Drugs/drug withdrawal
- Miscellaneous

TABLE 2: History and symptomology.

Symptoms and signs	• Light-headedness, nausea, vomiting • Drowsiness, mild confusion, hypotension, bradycardia, normal O_2 saturation
Which organ system should you explore?	Based on the symptoms above, systems including CNS/CVS/metabolic/inner ear/abdomen are the most appropriate to explore
Specific (succinct) history	• *CNS/inner ear:* Headache, facial droop, limb weakness, slurred speech, previous CVA/TIA, convulsions, vertigo, hearing loss, tinnitus. • *CVS:* Chest pain, shortness of breath, palpitations • *Abdomen:* Pain, diarrhea, PR bleed, vomiting (especially hematemesis) • Toxins (including alcohol)/infection (e.g., fevers)/trauma (especially head strike) *Patient reported vague chest pain for a few hours. No vertigo or tinnitus. Also, CNS and abdomen examinations were unremarkable*
Additional information (Dependent on the acuteness of situation)	• As the patient has responded yes to chest pain, cardiac and respiratory systems can be further explored • Site, onset, characteristics, at rest or exertion, radiation, previous history of IHD/stents, risk factors (HTN, DM, Fhx, hypercholesterolemia, and smoking), syncope/presyncope, orthopnea, PND, shortness of breath, calf pain, long haul flights, period of immobilization, OCP use (in a female), previous history of clots, productive cough, and fevers • Baseline function, past medical history (including cancer), medications

(CNS: central nervous system; CVA: cerebrovascular accident; CVS: cardiovascular system; DM: diabetes mellitus; HTN: hypertension; Fhx: family history of; IHD: ischemic heart disease; OCP: oral contraceptive pill; PND: paroxysmal nocturnal dyspnea; PR: per rectal; TIA: transient ischemic attack)

TABLE 3: Differential diagnosis.

Re-evaluation of differential diagnosis	• *Inner ear:* Meniere disease/labyrinthitis—will not cause hypotension • *CNS:* Ischemia to brain stem—will not cause hypotension • *CVS:* RV MI/inferior wall MI—can cause all above symptoms • *Abdomen:* Acute abdomen with dehydration causing shock—usually has tachycardia rather than bradycardia • *Renal failure:* Typically will not cause bradycardia, unless associated with hyperkalemia; however, this ECG is not typical of hyperkalemia The only condition in our list that will cause hypotension plus sinus bradycardia plus vomiting is *inferior wall MI*
Most likely clinical diagnosis	• Inferior wall MI • Others in our list less likely
Investigations	Instead of ordering every investigation under the sun, target them to the scenario • ECG as above—confirms ST elevation in inferior leads • Troponin raised • Echo—inferior wall and RV hypokinesis • As our most likely diagnosis was confirmed, no need for extensive tests for other differentials
Diagnosis	Inferior wall MI with cardiogenic shock from RV infarction and associated bradycardia

(CNS: central nervous system; CVS: cardiovascular system; ECG: electrocardiogram; MI: myocardial infarction; RV: right ventricular)

Examination

Focused Examination

This should occur simultaneously whilst organizing bedside investigations [such as electrocardiogram (ECG), blood gas, blood sugar level (BSL), and mobile chest X-ray (CXR)] with the help of your colleagues.

- Begin with ABCs—airway, breathing, circulation
- If ABCs are intact, only then move to assessing the patient otherwise advanced life support (ALS) algorithm should be followed.
- At the bedside, quickly assess for the following:
  - Body habitus, diaphoresis
  - Respiratory distress—use of accessory muscles and tachypnea
  - Higher mental function by using Glasgow Coma Scale (GCS)
  - Tissue perfusion using skin turgor, capillary refill time, peripheral temperature, cyanosis, and urine output
  - Pulse rhythm and character, jugular venous pressure (JVP), heart sounds, murmur, mean arterial pressure (MAP; if available), vasopressor requirement, lung fields, for example, presence of crackles, peripheral edema, fluid responsiveness (e.g., passive leg raise), bedside echo (cardiac tamponade, four-chamber apical view for wall motion abnormalities)
  - Abdominal distention, palpation for tenderness, or obvious masses

Findings: You find that your patient is a 65-year-old gentleman who is obese, in respiratory distress, diaphoretic, and clenching his chest. He is maintaining his airway, breathing spontaneously and has a pulse. He is confused and getting drowsier. His vitals indicate hypotension, bradycardia, and normal saturation. His peripheries are cool, cyanosed, and capillary refill time is >3 s. The JVP is not elevated, heart sounds are dual, and the chest is clear with nil peripheral edema.

Bedside investigations indicate that BSL is 5.3 mmol, normal CXR, and ECG **(Fig. 1)**.

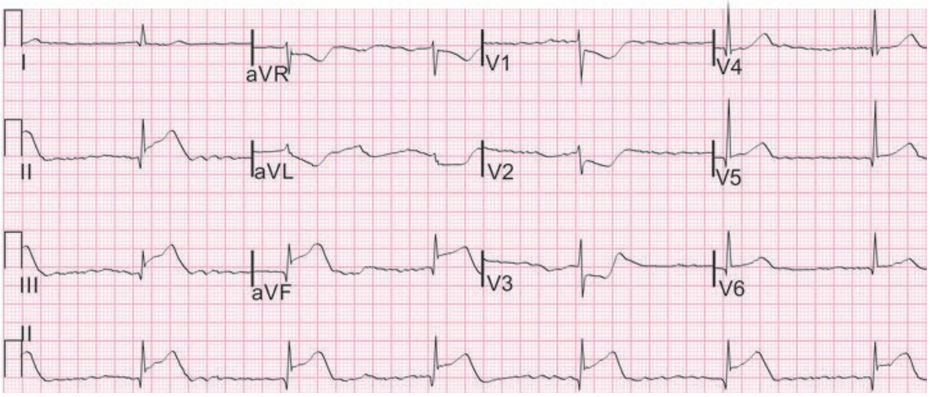

Fig. 1: ECG findings.
Source: Dr Smith's ECG Blog. (2013). Chest pain and Bradycardia. [online] Available from http://hqmeded-ecg.blogspot.com/2013/09/chest-pain-and-bradycardia.html [Last accessed April, 2024].

CASE SCENARIO 2

Symptoms	***Organ system and organ***	***Syndromic differential diagnoses***	***Specific history and pattern analysis***	***Likely clinical diagnoses***	***Specific investigations***	***Likely final diagnosis***
A 65-year-old woman with a history of type 2 diabetes mellitus presents with oliguric AKI. She has been generally unwell with no other specific symptoms for approximately 10 days	• Endocrine pancreas • Renal—AKI	• Unfortunately, these symptoms are much too nonspecific/vague for us to make any meaningful progress • We need more information • *Typically, we start with the vital signs:* – Deeply comatose – BP 139/73 mm Hg – Heart rate 76 beats/min (sinus) – O_2 saturation on room air = 100% – *ABG:* - pH 6.9 - paCO_2 54 mm Hg - Base excess = –19 mmol/L - Lactate = 17 mmol/L • *This additional data from the vital signs is quite helpful. Let us use the inverted pyramid approach (See Illustration 1 for details). I have presented it in a tabular format instead of a picture format that we used in case scenario 1. You may use whichever format you identify with*				
	CNS structural lesion	Ischemic/hemorrhagic CVA (stroke)	• Onset of coma was gradual (over days), not sudden/acute—this makes a stroke less likely, but still possible • No focal neurodeficits	Less likely, but still possible	CT brain normal—ischemic stroke less likely, but still possible; hemorrhagic stroke ruled out	• Hemorrhagic stroke ruled out • Ischemic stroke still possible, but less likely • Place this low in the list of differentials
	CNS nonstructural lesion	• Meningoencephalitis • Seizures	• No neck stiffness, but unreliable in the context of deep coma • No witnessed seizures, but unreliable	Less likely, but still possible	• No neck stiffness, but unreliable in the context of deep coma • No witnessed seizures, but unreliable • CSF unremarkable • EEG—no seizures; diffuse slow wave	Both meningo-encephalitis and seizures ruled out

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Symptoms	***Organ system and organ***	***Syndromic Differential Diagnoses***	***Specific History and Pattern Analysis***	***Likely clinical diagnoses***	***Specific Investigations***	***Likely final diagnosis***
	Non-CNS cause: Systemic causes	*Severe AKI-related complications:* • Uremic encephalopathy • Severe HAGMA	There are no specific findings for uremic encephalopathy or HAGMA	Both still possible	• Biochemistry confirmed severe rise in urea (27 mg/dL) and creatinine (3 mg/dL) • pH 6.9; paCO$_2$ 54 mm Hg • Base excess = –19 mmol/L • Lactate = 17 mmol/L • ABG confirmed severe metabolic acidosis with poor respiratory compensation	• Highly likely cause for coma = Uremic encephalopathy and severe HAGMA, with severe raised lactate • However, we still need to understand the cause of the AKI and lactate
		Sepsis/septic shock	• Normal BP and heart rate • No clinical focus of sepsis	This makes septic shock highly unlikely, but still possible	• Blood, urine, and CSF cultures—no growth • No sputum available • Chest imaging normal	• Septic shock ruled out as a cause for the deep coma, AKI, and lactate • Sepsis/infection still possible, so best to start empiric broad-spectrum antibiotics until things are clearer • Place this low in the list of differentials

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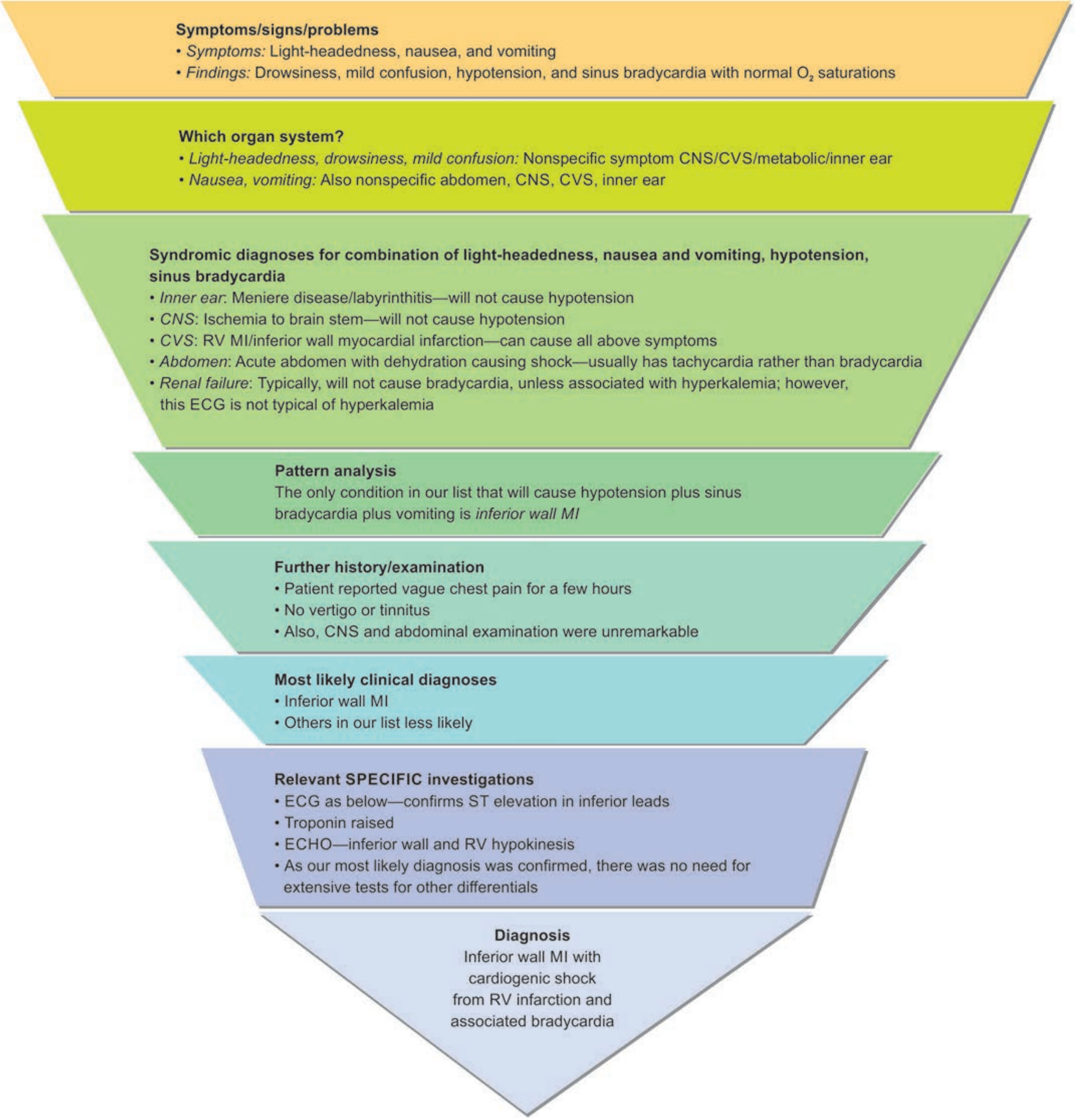
Symptoms	***Organ system and organ***	***Syndromic Differential Diagnoses***	***Specific History and Pattern Analysis***	***Likely clinical diagnoses***	***Specific Investigations***	***Likely final diagnosis***
		Fluid—dehydration	Family confirms history of recent gastroenteritis, diarrhea, and vomiting	Prerenal AKI highly likely	• Urine specific gravity 1.2 (concentrated) • Urine sodium 8 mmol/L—confirms prerenal state • CTKUB normal • Echo—small cardiac chambers with hyperdynamic contraction; small and collapsing IVC	Confirmed—Prerenal AKI from dehydration and hypovolemia
		Drug-induced coma	No past history of psychiatric illness/intentional/suicidal drug overdose	Unlikely but still possible	Urine drug screen normal	• Unlikely but still possible • Place this low in the list of differentials
			• Overdose/toxicity of prescription medications • *Family confirms the following medications:* – Telmisartan – Metformin – Aspirin – Atorvastatin	• This is very helpful to make a clinical diagnosis • In the setting of dehydration, telmisartan may have worsened the AKI • In a patient with AKI, metformin would cause severe lactic acidosis	No specific investigations. This remains a clinical diagnosis.	• Gastroenteritis → vomiting/diarrhea → dehydration → prerenal AKI • Additional nephrotoxic component from telmisartan • Metformin accumulation → lactic acidosis
		Electrolyte derangements—hypo/hypernatremia, hypo/hypercalcemia	Nothing specific apart from biochemistry	Nothing specific	• Serum Na = 149 mmol/L • Serum Ca = 1.1 mmol/L	Ruled out electrolyte derangement as a cause of the coma

(ABG: arterial blood gas; AKI: acute kidney injury; BP: blood pressure; CNS: central nervous system; CSF: cerebrospinal fluid; CTKUB: computed tomography kidneys, ureters, and bladder; CVA: cerebrovascular accident; EEG: electroencephalogram; HAGMA: high anion gap metabolic acidosis; IVC: inferior vena cava)



Symptoms/signs/problems

- *Symptoms:* Light-headedness, nausea, and vomiting
- *Findings:* Drowsiness, mild confusion, hypotension, and sinus bradycardia with normal O_2 saturations

Which organ system?

- *Light-headedness, drowsiness, mild confusion:* Nonspecific symptom CNS/CVS/metabolic/inner ear
- *Nausea, vomiting:* Also nonspecific abdomen, CNS, CVS, inner ear

Syndromic diagnoses for combination of light-headedness, nausea and vomiting, hypotension, sinus bradycardia

- *Inner ear*: Meniere disease/labyrinthitis—will not cause hypotension
- *CNS*: Ischemia to brain stem—will not cause hypotension
- *CVS*: RV MI/inferior wall myocardial infarction—can cause all above symptoms
- *Abdomen*: Acute abdomen with dehydration causing shock—usually has tachycardia rather than bradycardia
- *Renal failure*: Typically, will not cause bradycardia, unless associated with hyperkalemia; however, this ECG is not typical of hyperkalemia

Pattern analysis

The only condition in our list that will cause hypotension plus sinus bradycardia plus vomiting is *inferior wall MI*

Further history/examination

- Patient reported vague chest pain for a few hours
- No vertigo or tinnitus
- Also, CNS and abdominal examination were unremarkable

Most likely clinical diagnoses

- Inferior wall MI
- Others in our list less likely

Relevant SPECIFIC investigations

- ECG as below—confirms ST elevation in inferior leads
- Troponin raised
- ECHO—inferior wall and RV hypokinesis
- As our most likely diagnosis was confirmed, there was no need for extensive tests for other differentials

Diagnosis

Inferior wall MI with cardiogenic shock from RV infarction and associated bradycardia

Illustration 1: Inverted pyramid approach. Solving the Case Scenario 1 using inverted pyramid approach. (CNS: central nervous system; CVS: cardiovascualr system; ECG: electrocardiogram; ECHO: echocardiography; MI: myocardial infarction; O_2: oxygen; RV: right ventricle; ST: ST segment).

CASE SCENARIO 3

Symptoms	***Organ system and organ***	***Syndromic differential diagnoses***	***Specific history and pattern analysis***	***Likely clinical diagnoses***	***Specific investigations***	***Likely final diagnosis***
A 33-year-old woman with no previous comorbid conditions presents with a 1 week history of generalized unwellness. She reports easy bruisability over the past 48 hours	• History of generalized unwellness—too nonspecific, hence more information required • Easy bruisability over the past 48 hours—this is more helpful; it points to a bleeding disorder (thrombocytopenia/coagulopathy)	• We need more information • Typically, we start with the vital signs – Fully conscious and oriented—unlikely to be neurological – BP 120/78 mm Hg, heart rate 146 beats/min (sinus) - Hemodynamically unstable - However, the combination of normal consciousness and normal BP suggests that she is not shocked. – Temperature = 39°C (102.2°F) - Infective versus noninfective - Thorough organ system-focused history and physical examination to identify source – O_2 saturation on room air = 100%—unlikely to be respiratory focus • The junior doctor made a provisional diagnosis of septic shock with multiorgan failure based on some lower lobe mild infiltrates on CXR. – *At one level, it is reasonable to consider septic shock as the top differential, based on the constellation of fever, tachycardia, CXR changes, and bruisability. However, there are two unresolved issues.* - *One, the patient does not appear to be shocked, based on her normal sensorium and BP. However, compensated shock is still possible.* - *Two, the mild CXR infiltration and normal O_2 sats are out of keeping with the degree of fever and tachycardia.* – *Hence, we need to use the inverted pyramid approach to start broad and systematically narrowing down on the possibilities.*				
	CNS structural lesion	Ischemic/hemorrhagic CVA (stroke)	• Sensorium normal • No focal neurodeficits	Unlikely	Not needed	Unlikely to be an issue
	CNS nonstructural lesion	• Meningoencephalitis • Seizures	• Sensorium normal • No focal neurodeficits • No neck stiffness/seizures	Unlikely	Not needed	Unlikely to be an issue

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Symptoms	**Organ system and organ**	**Syndromic differential diagnoses**	**Specific history and pattern analysis**	**Likely clinical diagnoses**	**Specific investigations**	**Likely final diagnosis**
	Non-CNS cause: Systemic causes	Septic shock	• Organ-focused history and physical examination required • *Bruisability:* – Check for petechiae (platelet disorder) versus purpura (coagulopathy)	Findings show diffuse petechial hemorrhages—thrombocytopenia is likely • ?cause • Follow your approach for thrombocytopenia	• Cultures negative • Complete blood count confirmed severe thrombocytopenia and anemia with normal WBC count • Further investigations required.	• Although septic shock is possible, it is progressively becoming less likely, given lack of clinical focus and negative cultures • Noninfective systemic inflammation such as autoimmune causes are becoming more likely • *Consider TTP/HUS/HLH as differentials:* – Blood film showed schistocytes confirming hemolysis – Send ADAMSTS13 – Kidney function normal—unlikely HUS • Ferritin 30,000—HLH likely • However, it is imperative to continue evaluating and empirically treating for a source of sepsis

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Symptoms	**Organ system and organ**	**Syndromic differential diagnoses**	**Specific history and pattern analysis**	**Likely clinical diagnoses**	**Specific investigations**	**Likely final diagnosis**
			• *Additional symptoms:* – Vague *abdominal pain:* - Examine using the approach for abdominal pain - Examination → Mild tenderness in RUQ, but no peritonism; no murphy • Now, do a focused evaluation for bowel – No other bowel symptoms (vomiting, diarrhea, constipation) • *Do a focused evaluation for hepatitis:* – Mild scleral icterus on examination—liver derangement likely – Evaluate history and examination for hepatotoxins, alcohol, gallstones, right heart congestion, etc.	• Low likelihood of cholecystitis (absent Murphy sign) • Hence, RUQ pain likely to be hepatitis • More examination and investigations evaluation required for this	• *All LFTs moderately raised:* – Transaminases, GGT, and ALP mildly raised to ~300s – Bilirubin mildly raised – Indicates mixed picture • Although we had clinically ruled out biliary sepsis before, it is important to continue investigate and rule it out as a cause of the mixed LFT rise and fever • Biliary ultrasound did not show cholecystitis or cholangitis	• Nonspecific hepatitis as a part of a systemic inflammation • Based on all this and the high ferritin, the patient had a bone marrow biopsy. This confirmed HLH. She improved with immunosuppressive therapy with glucocorticoids

(ALP: alkaline phosphatase; BP: blood pressure; CNS: central nervous system; CVA: cerebrovascular accident; CXR: chest X-ray; GGP: gamma-glutamyl transpeptidase; HLH: hemophagocytic lymphohistiocytosis; HUS: hemolytic uremic syndrome; LFT: liver function test; RUQ: right upper quadrant; TTP: thrombotic thrombocytopenic purpura; WBC: white blood cell)

SEQUENTIAL APPROACH TO SYNDROMIC DIAGNOSIS

Step 1: Identify the systems potentially affected.

Step 2: Get the system-based diagnosis, that is, for each organ system arrange the possible diagnosis based on the available information.

Step 3: Gather more information to add to the existing information (history, examination, and POCUS).

Step 4: Pattern analysis to narrow down the diagnosis.

Step 5: Appropriate investigations to confirm the diagnosis.

Step 6: Confirmation of diagnosis.

SUMMARY

- Start broad and go narrow.
- Order your working diagnosis as likely or unlikely using history, examination, and investigations.
- Have a list of common pathologies pertaining to each body system.
- ABCs first—stabilizing the patient first is essential in acute life-threatening settings before forming a diagnosis.

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Chapter

7

Identifying the Impending Signs of Organ Failure

Subhash K Todi

"Organ failure does not happen suddenly, it is detected suddenly."
"Early sepsis is difficult to diagnose but easy to treat, late sepsis is easy to diagnose but difficult to treat."

ABSTRACT

Time is essence in critical care. Physical examination is the quickest way to detect deteriorating physiology of the patient. Early warning signs have been described to identify this and initiate a rapid response team. A systematic approach to a physical examination is needed to identify conditions requiring immediate interventions. In critically ill history taking, physical examination and resuscitation has to be taken simultaneously rather than sequentially. Serial physical examination needs to be conducted to identify features which are missed during initial examination or any new signs which might have developed later.

CASE SCENARIO

An 80-year-old male hypertensive with a history of dementia was admitted to the ward with fever and dysuria. The nurse noticed him to be restless and confused, his pulse rate was 90 beats/min regular, BP was 130/80 mm Hg, respiratory rate was 24 breaths/min, temperature 100°F, and oxygen saturation 96% on ambient air. He passed 300 mL of urine in the last 6 hours. You are asked to see him. You are wondering whether he is confused because of dementia or his infection is worsening and he is getting septic and developing septic encephalopathy. You have to make a decision as to whether to transfer him to the intensive care unit.

INTRODUCTION

The scenarios as the one mentioned above are encountered frequently in hospital practice. Identifying organ deterioration early will enable the physician to monitor the patient more closely, escalating any supportive therapy and, if available, offer any definitive treatment. This chapter will be a narrative review of the early detection of warning signs of deterioration in a hospitalized patient.

OBJECTIVE SCORING SYSTEMS

Early Warning Scores

Deterioration in vital signs reflecting abnormal physiology is the earliest sign of impending critical illness and organ dysfunction. These are easily detectable, measurable, reliable, actionable, and inexpensive. A combination of ≥1 of these vital signs with different cutoffs has been validated over years in different patient population. They constitute the "afferent arm" of "track and trigger" strategy of rapid response system. The efficacy of rapid response system to ultimately change patient outcome may be debatable, but it is undeniable that the early warning signs utilized in triggering the response are able to detect early the patient population who are likely to deteriorate. Some of the commonly used early warning systems (EWS) with their details are described below. These EWS should be differentiated from severity scoring system such as Acute Physiology and Chronic Health (APACHE), morbidity and mortality prediction models (MPM), etc. The severity scoring systems are generated after a certain prespecified time such as 24 hours in APACHE in a specified patient population, they contain both clinical and laboratory parameters, and take into account patients underlying diagnosis and comorbidities. Though they predict hospital mortality, they are not useful for prediction on an individual patient but are applicable in a group of similar patients so as to compare performance measures. EWS on the other hand can be applied to any patient population and is more of a triage decision tool of that individual patient.

- *Modified Early Warning Score (MEWS)* ***(Table 1)****:* This was the first composite EWS to be developed in 1997 and validated in a hospitalized medical patient in 2001. This score is a composite of systolic blood pressure, heart rate, respiratory rate, body temperature, urine output, and conscious level, which is assessed as alert, response to voice, response to pain, and unresponsiveness (AVPU). Variable risk scores were given to values on either side of the normal physiological values. Score >5 was associated with five times more risk of death, thus it indirectly estimates the risk of impending organ failure. Authors advocated a creation of clinical pathway based on the score for proper identification and triaging of patients depending on the MEWS score. In another study, a combination of clinical judgment with MEWS score of ≥4 was more sensitive to detect a deteriorating patient, asserting the fact that by using clinical judgment alone we might be overestimating the risk of clinical deterioration. In the clinical example cited above, the MEWS score would be respiratory rate: 2, systolic blood pressure: 0, heart rate: 0, urine output: 0, and neuro: 2. Composite score of 4 does not put him into high risk of deterioration.
- *National Early Warning System (NEWS)* ***(Table 2)****:* This was developed by National Health Services United Kingdom to standardize EWS throughout the country. It is similar to MEWS, but removes urine output, which is sometimes difficult to ascertain outside intensive care unit (ICU), and adds oxygen saturation and need for supplemental oxygen. The frequency with which this score is to be calculated is variable, but at least once every 12 hours and more frequently if score worsens. An aggregate score of >5 or >3 in any one parameter warrants an urgent assessment of patient. Application

TABLE 1: Modified Early Warning Score.

Score	***3***	***2***	***1***	***0***	***1***	***2***	***3***
Respiratory rate (min^{-1})		≤8		9–14	15–20	21–29	>29
Heart rate (min^{-1})		≤40	41–50	51–100	101–110	111–129	>129
Systolic BP (mm Hg)	≤70	71–80	81–100	101–199		≥200	
Urine output (mL/kg/h)	Nil	<0.5					
Temperature (°C)		≤35	35.1–36	36.1–38	38.1–38.5	≥38.6	
Neurological				Alert	Reacting to voice	Reacting to pain	Unresponsive

(BP: blood pressure)

TABLE 2: National Early Warning System (NEWS).

Score	***3***	***2***	***1***	***0***	***1***	***2***	***3***
Respiratory rate (breaths/min)	≤8		9–11	12–20		21–24	≥25
O_2 saturation (%)	≤91	92–93	94–95	≥96			
Added O_2		Yes		No			
Systolic BP (mm Hg)	≤90	91–100	101–110	111–219			≥220
Heart rate (bpm)	≤40		41–50	51–90	91–110	111–130	≥131
Level of consciousness				A			V.P.U
Temperature (°C)	≤35.0		35.1–36.0	36.1–38.0	38.1–39.0	≥39.1	

(A: alert; BP: blood pressure; P: responds to pain only; U: unresponsive to stimuli; V: responds to verbal stimuli. In the NEWS, scoring missing values are interpreted as normal values) of NEWS to prevent cardiac arrest in the floor had variable degree of efficacy. Further modification of NEWS was made to stratify oxygen saturation criteria in hypercapnic versus nonhypercapnic patient and adding "confusion" to neurological assessment. In clinical practice though NEWS2 did not had any extra advantage over NEWS.

- *Cardiac Arrest Risk Triage (CART) score:* A simpler early recognition tool was developed and validated with easily measurable vital signs. The vital signs included respiratory rate, heart rate, diastolic blood pressure, and age. The score compared favorably with MEWS score to predict early deterioration of the patient.
- *Quick Sequential Organ Failure Assessment (qSOFA):* In 2016, a new definition of sepsis, Sepsis-3 was proposed by the international task force. The task force coined the term qSOFA or quick SOFA to identify patients early who are the risk of dying from sepsis. These criteria consist of respiratory rate, systolic blood pressure, and mental state **(Table 3)**. A score of ≥2 indicates organ dysfunction.
- *Shock index (SI):* Heart rate and blood pressure are universal measurements in ICU patients, but they are often interpreted separately. A composite index of SI is defined as the heart rate divided by systolic blood pressure may be more important in identifying a deteriorating patient before both of the components separately become abnormal. In a study, patients with an SI ≥1.00 from initial emergency department (ED) vital signs correlated with a higher mortality rate.
- *Diastolic shock index (DSI):* After initial fluid resuscitation, sepsis usually manifests as a vasodilatory state with a low diastolic pressure due to decreased vascular tone. Moreover, there is a correlation of diastolic pressure with the heart rate as in normal state with tachycardia, the diastolic time decreases and the diastolic pressure does not get time to decrease much. Thus having a low diastolic pressure in spite of tachycardia is a bad prognosis sign. Based on this observation, DSI has been defined as the ratio between heart rate and diastolic blood pressure and increasing DSI is a marker of bad outcome. Systolic SI may be more appropriate for absolute hypovolemic state as may be seen in early sepsis, whereas DSI may be more appropriate at a later phase of sepsis with relative hypovolemia and vasodilatation.

TABLE 3: Quick Sequential Organ Failure Assessment (qSOFA) score.

qSOFA criteria	*Points*
Respiratory rate ≥22 breaths/min	1
Change in mental status	1
Systolic blood pressure ≤100 mm Hg	1

Clinical Examination

- *Capillary refill time (CRT):* Whenever there is flow limitation due to hypoperfusion, the first defense of the body is to divert flow from nonessential organs to essential organs. Skin perfusion suffers initially and can be early warning feature of impending organ failure **(Box 1)**. Efforts have been made to standardize the procedure. It is defined as the time taken for distal capillary bed to regain its perfusion after pressure has been applied to it to cause blanching. It is usually performed by pressing the distal nail bed or pulp for 3 seconds or till it blanches and release it and measure the time in seconds for the area to regain its color. One usually needs a stopwatch to time it correctly and sometimes may require two persons, one for observation and another for timing, which becomes cumbersome in clinical practice. Another way of overcoming it is to time the return by counting "1 in 100," "2 in 100," and so on till the color returns. Each count is equivalent to 1 second and should be practiced by the observer beforehand. Normal CRT is <3 seconds, but it varies with age and may be a little higher in elderly. CRT observation may become confounded by dark skin color and nail bed may be preferred than pulp of finger. Recently, a randomized study has been conducted comparing CRT with lactate as endpoint of resuscitation in septic shock and CRT was found to be noninferior to lactate. Based on this and other observational studies, the latest surviving

BOX 1: Impending signs of organ failure.

- *Observe for subjective features:* Delirium, anxiety, and facial expression
- *Examine clinically for global features of hypoperfusion:* Capillary refill time and cold clammy skin
- *Examine for organ-specific features of impairment:* Glasgow Coma Scale, accessory muscle use, oliguria, etc.
- *Use early warning scores from vital signs:* NEWS, MEWS, and qSOFA

(MEWS: Modified Early Warning Score; NEWS: National Early Warning System; qSOFA: Quick Sequential Organ Failure Assessment) sepsis campaign iteration has recommended CRT measurement as an adjunctive measure to detect hypoperfusion. CRT becomes more important in resource-limited settings where other advanced tools for hemodynamic monitoring are not available and should be routinely used by all clinical practitioners as a part of their assessment of severity of patients' perfusion status, which may ultimately lead to organ failure. CRT not only helps in early detection of hypoperfusion but also is a tool to guide initial resuscitation and fluid therapy in shock states.

- *Skin temperature:* With the flow diversion from nonessential organs such as skin to essential organs during flow limitation states, there is a proportional decrease in skin temperature and just like CRT, this may be a guide to estimate decreased perfusion state, which if prolonged can lead to organ failure. Moreover, the core temperature is maintained, thus the difference between core and peripheral temperature gets widened and may be the earliest indication of flow reduction and impending organ dysfunction. Skin temperature may be measured subjectively by touch or palpation or by temperature measuring devices. Core temperature such as tympanic, rectal, and bladder temperature can be measured by various devices.
- *Organ-specific:* Apart from the clinical examination, which indicates global perfusion status, there are organ-specific early findings that may reflect the impending failure of the organ concerned.
  - *Neurological:* Glasgow Coma Scale and FOUR Score
  - *Respiratory:* Tachypnea, accessory muscles use, and dyspnea
  - *Cardiac:* Sweating and chest discomfort
  - *Abdominal:* Pain and tense abdomen intolerance to feed
  - *Renal:* Decreasing urine output and increasing acidosis

SUBJECTIVE IMPRESSION

- *Anxiety:* A higher anxiety level as assessed by subjective criteria in patients requiring intensive care admission has been associated with new organ failure independent of the severity of illness and respiratory status at admission. This association has been found in a prospective study but does not imply causality.
- *Facial expression:* As the patient is approached, one should stand at the bedside and just observe the patient for a few seconds. Facial expressions may reveal features of distress, which could be the earliest sign of impending organ dysfunction even before the objective vital signs become abnormal. Patterned facial expressions were identified and correlated with NEWS score. Making oneself familiar with these patterns may help in closely monitoring such patients. A time series analysis showed patients admitted to intensive care have a decrease in the number of facial expressions per unit of time and an increase in their diversity.
- *Detecting delirium early:* When approaching a patient and querying about their health status in ICU, the answer "I am perfectly normal" may be a subtle feature of early delirium. Delirium is usually accompanied by worse outcomes in ICU and organ dysfunction. With the advent of wearable sensor technology, it was demonstrated that by monitoring patients' faces, their postures, facial action units, expressions, head pose variation, extremity movements, etc., delirium could be detected early in these patients.

SUMMARY

In the case scenario presented in the introductory section, most of the vital signs of the patient are normal, except for increased respiratory rate and delirious state, which may reflect an early onset of sepsis. A quick sepsis workup, including cultures, etc., needs to be sent and an empirical antibiotic should be started along with close monitoring to avoid impending organ dysfunction.

Impending signs of organ failure may be judged both by objective and subjective means. While the objective scoring system is useful in identifying patients early for deterioration and helps in triaging, robust evidence from randomized studies are lacking as to whether it improves clinically meaningful outcome. One of the reason for this paradox could be that the underlying disease ultimately determines outcome and early detection only changes the setting of ultimate outcome, that is, from ward to ICU. Judicious fluid management and safe ventilation should be practiced irrespective of whether there are features of impending organ dysfunction. Apart from being more vigilant and placing the patient in an appropriate monitoring environment, at present we do not have much therapeutic options to specifically prevent organ dysfunction with organ protective drugs. Nevertheless, exploring features of impending organ dysfunction will help us in applying the tools of organ protection once they become available.

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SECTION 2

Systemic Examination

8. **Central Nervous System Examination in a Critically Ill Patient: Part 1 Brain**
George John

9. **Central Nervous System Examination in a Critically Ill Patient: Part 2 Neuromuscular System**
Farhad N Kapadia, Jare Jagannath Uddhavrao

10. **Respiratory System Examination in a Critically Ill Patient**
Jose Chacko

11. **Cardiovascular System Examination in a Critically Ill Patient**
Ram E Rajagopalan

12. **Gastrointestinal System Examination in a Critically Ill Patient**
Raghavendra Kotal, Pradeep Rangappa

13. **Examination of a Critically Ill Immunocompromised Patient**
Sunil Karanth, Raghavendra B Goudar

Chapter

8

Central Nervous System Examination in a Critically Ill Patient: Part 1 Brain

George John

ABSTRACT

Clinical examination of the brain is an essential component of the clinical evaluation of a critically ill patient. It should be done systematically taking into consideration the constraints of examination in a critically ill patient in an intensive care unit setting.

CASE SCENARIO 1

A 20-year-old girl from a village was brought to the emergency department of a tertiary care center with a history of being unarousable after going to sleep on the verandah of her house in the village. There was no history of ingestion of any poison. The patient was deeply cyanosed, had total ophthalmoplegia, dilated pupils, and absent pupillary, vestibulo-ocular, and corneal reflexes. There were no facial movements or limb movements in response to pain. Deep tendon reflexes were not elicitable.

INTRODUCTION

The most important organ which is being supported in any intensive care unit (ICU) is the brain. To give an analogy, in a chess game, if the king is lost, the game is lost, even if all the other pieces are on the board. Similarly, in any clinical scenario, if a person's brain is irreversibly damaged, all is lost in terms of the meaningful survival of the person.

This section will focus on the examination of the brain and cranial nerves relevant to the ICU patient. In this context, the evaluation of focal cortical deficits such as aphasia, agnosia, or apraxia are only discussed in so far as that it affects the performance of the clinical examination and its interpretation.

The clinical neurological evaluation, to be useful, must be done meaningfully (understanding what is being tested) rather than mechanically. To obtain meaningful information, it must be systematically done and documented. Performing a minutely detailed neurological examination without a purpose is a waste of time and can yield minor findings, which are red herrings and cloud the picture.

All details of scoring systems in this discussion are given at the end of the section to preserve the continuity of one's flow of thought while reading.

The key to neurological evaluation is the *observation*.

- *Presence or absence* of a response—*spontaneous* or *induced* with a stimulus
- *Compare: Asymmetry* of a response is the key to recognizing subtle and early signs—see both sides; observe for unilateral/asymmetrical response
- *Serial evaluations over time* are essential to recognize trends and pick up early deterioration
- *Confounders* such as sedation, analgesia, neuromuscular paralyzing agents, metabolic disturbances, and *constraints* to examination such as injuries, intubation, and bandages must be factored in the assessment.

Patients in an ICU with a neurological problem may have the following scenarios:

- Admitted with a specific critical neurological problem:
    - Depressed sensorium/coma
    - Cerebrovascular accident
    - Head injury
    - Postcardiac arrest
    - Seizures
- Develop a neurological problem as a result of being critically ill due to other organ system failures:
    - Delirium
    - Critical illness neuromuscular abnormalities

Examination of the brain and cranial nerves is important in all the above scenarios for:

- *Diagnosis:*
  - What is the lesion?
  - Where is the lesion?
  - Brain death: change the focus of support
    - Withdrawing care
    - Organ donation/harvesting
- *Prognosis:*
  - Is ICU care likely to be beneficial?
  - Family counseling regarding cost, palliative mode of care, and long-term care
- *Change in status recognition/documentation:*
  - Monitor and document any improvement or deterioration

Basic rules:

- *Restlessness in any critically ill patient should be assumed to be due to cerebral hypoxia unless proven otherwise. Rule it out before proceeding to a formal evaluation.*
- *Altered sensorium in the ICU may be medication related: Check the medications before interpretation.*

The examination of the brain and cranial nerves should be systematic and sequential. A common template that can be followed is to assess the brain function into the following subdivisions:

- Consciousness
- Thought content
- Cranial nerves

ASSESSMENT OF CONSCIOUSNESS

The assessment of consciousness may differ depending on the patient's status:

- *Altered sensorium:*
  - What is the *level* of consciousness?
  - What is the *content* of consciousness?
- *Normal sensorium:*
  - Focal deficits with impaired communication: *Aphasia/agnosia/apraxia*
  - Focal deficits with normal communication: *What and where is the lesion?*

Patients with Altered Sensorium

There are two functioning neurological structures needed for consciousness—*the brain stem* and *one cerebral cortex*.

Consciousness has two components:

1. *Arousal* (wakefulness, level of consciousness) is the *overall level of alertness* to environmental stimuli (*wakefulness*). Arousal is a set of changes that occurs when the person transits to alertness—the most obvious sign being the opening of the eyes. It depends on an intact, functioning ascending reticular activating system (ARAS) passing through the *thalamus* to the *cerebral cortices*. It is a subcortical phenomenon and needs a *functioning brainstem*. *Bilateral thalamic lesions* can also cause coma because they interrupt the ARAS fibers passing through them to the cortex **(Table 1)**.

   The capacity of lesions in the brainstem to cause coma depends on its site, size, and speed of progression. Brainstem infarctions and hemorrhage commonly cause coma, whereas slower lesions such as multiple sclerosis or slowly infiltrating neoplasms rarely do so. *Lesions below the pons rarely cause coma.*
2. *Awareness* is the ability to perceive specific environmental stimuli in different visual, auditory, and somatosensory domains. It is the ability to be aware of one's environment and understand one's relationship to one's surroundings (*responsiveness*). This depends on the *content* of consciousness and is the summation of a person's cognitive and affective (emotional) mental functions. *Awareness* (the content of consciousness) depends on the integrity of the *cortex* and the *limbic* system (basal forebrain, amygdala, hippocampus, hypothalamus, and cingulum; **Table 1**).

In a nutshell, the state of arousal is easily confirmed by the opening of the eyes, and awareness is inferred from the ability to follow commands.

TABLE 1: Classification of conscious states.

Aroused, aware	***Aroused, unaware***	***Unaroused, unaware***
Normal	Delirium: hypoactive, hyperactive, mixed	Sleep, deep sedation
Anxiety, depression	Psychosis, dementia	Coma
Lethargy	Vegetative state	Brain dead

Comatose Patient

The cortex of a comatose patient is, by definition, dysfunctional. This may be either primary or secondary because of a dysfunctional ARAS. Drugs and metabolic states induce coma by depressing both the ARAS and the cerebral cortex.

If the brainstem function is intact, then the coma is due to a *primary cerebral cortical dysfunction*. If one or more levels of the brainstem are damaged, then the *cerebral cortical dysfunction* is *secondary*, and the cause of coma is at the brainstem level.

Examination of a comatose patient is first focused on assessing the brainstem function. This is carried out in a craniocaudal format, level by level. A *rapid 4-layer functional examination* and interpretation is useful in the ICU. Level-by-level testing of the brainstem involves testing the brainstem at each level with at least one neurological reflex. If the response at a particular level is abnormal, the corresponding brainstem level is considered dysfunctional.

If all 4 levels of the brainstem are functioning normally, then the coma is cortical (the cortical dysfunction is primary). *A unilateral primary cortical dysfunction does not cause coma unless there is pre-existent dysfunction in the other cortex or there is a secondary brain stem dysfunction (herniation).*

Levels

First step: Response to verbal instructions

Instruct the patient to open the eyes and look up and down and side to side; if the patient opens the eyes and follows all verbal commands appropriately as requested, the *level and content* of consciousness are *normal*.

If the response is not as expected, check the following described further.

Level 1: Thalamus—Response to Pain

- *Stimulus site:* Supraorbital, nail bed, and sternum
- *Observe:* Presence of response, nature of the response, and symmetry of response
- *Appropriate:* Push away stimulus or withdraw from it
- *Inappropriate responses and interpretation:*
  - *Decorticate:* Flexion of upper limbs and extension with internal rotation of lower limbs
    - *Interpretation:* Subcortical/thalamic dysfunction
  - *Decerebrate:* Extension and internal rotation of both upper and lower limbs
    - *Interpretation: Upper brainstem (midbrain or upper pons) dysfunction*
  - No response or extension of the upper limbs, bending of knees
    - *Interpretation:* Reflex response at the spinal level.

Level 2: Midbrain—Pupillary Reflex

- *Stimulus:* Light is shown into the pupil and response is observed; one can use an ophthalmoscope to magnify the pupil and see minimal responses.
- *Observe:* Pupillary constriction on both ipsilateral and contralateral pupils in response to light
- *Appropriate:* Details are given in the section on the examination of cranial nerves under ocular nerves.
- *Inappropriate responses and interpretation: Lesions above the thalamus and below the pons spare pupillary responses*

Level 3: Pons

- *Doll's eye reflex (oculocephalic/oculovestibular reflex)*
- *Corneal reflex*
- *Respiratory pattern (pons and medulla)*

If cervical spine injury is suspected, do not turn your head.

Details are given in the section on the examination of cranial nerves under ocular and trigeminal nerves.

Level 4: Medulla

Spontaneous cardiorespiratory changes:

- Apnea
- Unstable blood pressure
- Irregularities in heart rate and rhythm.

Coma Look-Alikes (Differential Diagnosis)

Locked-in Syndrome

This is due to a focal brainstem lesion usually involving the junction between the upper third and lower two-thirds of the pons. There is enough ARAS in the upper third of the pons to *preserve wakefulness and content of consciousness*. However, all functions below the pontine level are lost. These patients can move muscles controlled by cranial nerves III and VI—ocular movements are preserved but extremities cannot be moved. They can see and hear the examiner but sense neither touch nor pain. The eyes are open (in a true coma they are closed) and they can track the examiner. They may communicate using eye movement.

Vegetative State

These patients can be aroused (wakefulness) but lack content of consciousness. There is *awareness-arousal dissociation*. It has been named by some as a *"locked-out" syndrome* in that the cortex is unaware of the external world and so is disconnected from it. This can be due to widespread cerebral cortical damage (hypoxia or ischemia), diffuse damage to subcortical structures, or bilateral thalamic damage.

Psychological Dysfunctions

Hysteric (Conversion) reaction: This can mimic coma. One common clinical test used is to raise the upper limb above the face and let it drop. In conversion reaction, the face will be protected by the patient by allowing the upper limb to fall slowly to the side.

Catatonia: The patient is awake; the eyes are open but immobile. Transient improvement with sedation may occur.

The *level of consciousness* can be objectively assessed and documented by *any* of the following methods:

- *Alert, Voice, Pain, Unresponsive (AVPU) score*
- *Full Outline of Unresponsiveness (FOUR)*
- *Glasgow Coma Scale (GCS) score*
- *Richmond Agitation Sedation Scale (RASS)*
- *Ramsay Sedation Scale (RSS)*

Noncomatose Patient

Mentation, Emotions, Thought Content: Remember the acronym—*EMOJI*

- *Emotions:* Observe and evaluate whether the emotions exhibited are *appropriate/inappropriate and if appropriate, proportionate* to the situation.
- *Memory: Immediate, recent,* and *remote* memory can be tested
- *Orientation:* Tested with reference to *time, place, and person*
- *Judgment:* Ability to make a judgment in ambiguous situations
- *Intellect:* Test with spelling 5 letters *words* forward or back, serial *calculation* backward or forward.

The following conditions can interfere with neurological evaluation but are not discussed further:

- *Aphasia:* Acquired disorder in *using language* due to a lesion in the *dominant cerebral cortex*.
- *Apraxia:* Difficulty in performing a *complex motor task* despite normal primary motor functions. It occurs due to lesions in the *association areas* of the *frontal cortex* in either *dominant* or *nondominant hemispheres*.
- *Agnosia:* Difficulty in recognizing a *complex sensory stimulus* in the presence of normal primary sensations. It occurs due to lesions in the *association cortex in either dominant* or *nondominant hemispheres* in the *parietal* or *temporal cortices*.

THOUGHT CONTENT

Delirium is an *acute disturbance of cognition* that develops over a short timeline. It manifests itself by a clouding of consciousness (decreased clarity of awareness of the environment) and fluctuates with time. It manifests itself as confusion in the patient's thought process. Its characteristic time profile is its *acute onset* and *fluctuating clinical course*. It can manifest in the following three forms:

1. *Hypoactive:* Lethargic, decreased alertness and activity
2. *Hyperactive:* Restless, agitated, pull out tubes and lines
3. *Mixed:* Fluctuates between the two states

Validated instruments to identify delirium in the ICU are available. The two which are commonly used are as follows:

1. *Confusion Assessment Method for the ICU (CAM-ICU)*
2. *Intensive Care Delirium Screening Checklist (ICDSC)*

These scores can only be used if the patient is not comatose (RASS score must be ≥ -3*).*

CRANIAL NERVES

The clinical evaluation of the 12 cranial nerves is done in sequential order. In critically ill patients, it may not be possible to test all of them in every patient. Sensory components of the cranial nerves are difficult to interpret in those who are not fully conscious, but a normal reflex implies that the sensory component is intact. The technique of testing, interpretation of the response, its significance in the critically ill patient, and the constraints to testing are discussed here.

- Olfactory
- Optic
- *Ocular nerves:* Oculomotor, trochlear, and abducens
- Trigeminal
- Facial
- Vestibulocochlear
- Glossopharyngeal, vagus
- Accessory
- Hypoglossal

Most cranial nerves are *bilaterally controlled* via *upper motor neurons (UMNs)* originating in *both cerebral cortices*. The notable exceptions are:

- Lower part of facial nerve nucleus (only *contralateral* UMN control in *all* persons)
- Trigeminal and hypoglossal (only *contralateral* UMN control in *some* individuals)

Olfactory Nerve: I

The sensation of smell can be tested in each nostril using familiar substances (coffee powder). However, diminished or altered sensorium, presence of a nasogastric (NG) tube, or blood in the nasal cavity preclude testing. Testing should not be done with irritant substances because they stimulate the pain fibers of the trigeminal sensory component—the pain sensation is being tested, not olfaction. This fact is useful and can be used to test the olfaction of a person who is suspected to be malingering, stating that he/she has lost olfaction after trauma. The olfactory fibers traverse the cribriform plate of the ethmoid bone and can be genuinely damaged in head trauma. However, in a malingering patient, both sensation of smell and noxious substances when tested are stated to be lost—though they traverse different pathways. This has medicolegal consequences with respect to compensation.

Optic Nerve: II

The testing components are:

- Visual acuity
- Visual fields
- Color vision
- Pupillary reflexes
  - Light
  - Accommodation

In the ICU, vision can be grossly tested by the *menace response*. The menace response involves cerebral cortical integration and is not a reflex response. This test evaluates the behavioral response to a visual threat. It is a cortically mediated closure of both eyelids produced by a threatening or unexpected gesture or action suddenly appearing in the near visual field. The response consists of the afferent pathway (optic nerve, optic tract, lateral geniculate body, and optic radiation), occipital cortex (integration), and facial nerve (efferent). Care must be taken not to create too much air turbulence, which will elicit the corneal reflex. One eye is covered, and a hand is then moved rapidly toward the open eye stopping prior to causing physical contact. The normal response seen is the reflex bilateral blinking that occurs in response to the rapid approach of an object. The test is then repeated for the other eye.

Visual field and color vision cannot be adequately tested in the ICU and are not useful. The pupillary reflex (especially the light reflex) is very important and is given in detail in the section on ocular nerves.

Ocular Nerves: III, IV, VI

The following are important to observe:

- Eyelids
- Eye movements
- Pupils

1. The oculomotor nerve has two nuclei in the midbrain:
   a. *Oculomotor nucleus (somatic and motor)*
   b. *Edinger-Westphal nucleus (parasympathetic nucleus for pupillary control)*
2. The trochlear nerve nucleus is also in the midbrain.
3. The abducens nerve nucleus is in the upper pons.

In a patient with orbital trauma, the findings are difficult to observe and interpret.

Eyelids

- Observe for ptosis and eye opening
- Neuromuscular causes of ptosis are as follows:
  - Oculomotor lesion
  - *Sympathetic lesion:* Horner syndrome
  - Muscle diseases, for example, myasthenia (usually bilateral)

Eye Movements

Remember that the contralateral trochlear nerve [lower motor neuron (LMN)] innervates the eye being examined (in contrast to all the other cranial nerves where the ipsilateral cranial nerve lower motor neuron innervates the relevant muscles) **(Figs. 1A and B)**.

In a person with delirium, eye movements *cannot be tested meaningfully*.

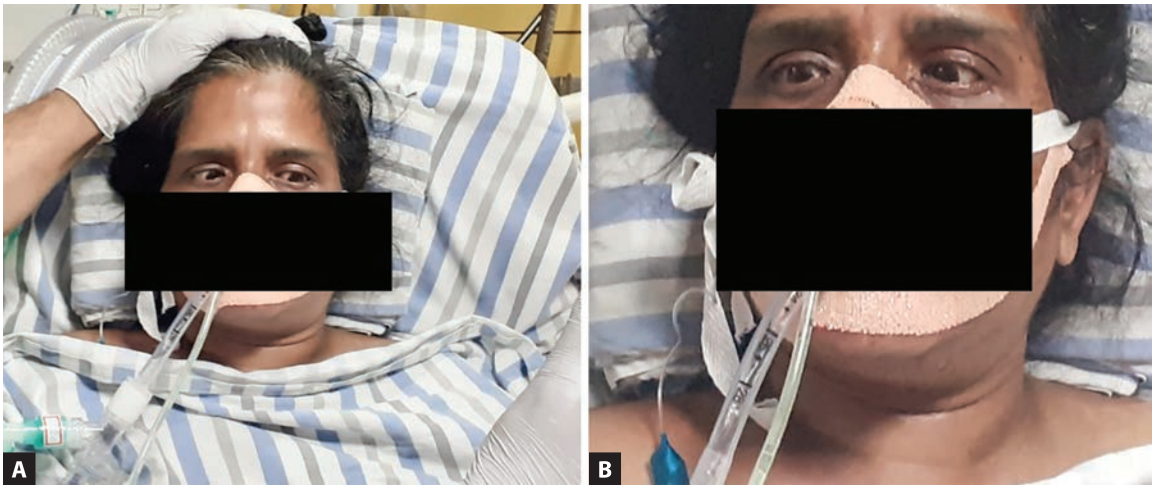
In an *unconscious patient*, the *oculocephalic* and *oculovestibular reflexes* are tested to evaluate the integrity of the vestibular system, brain stem, and ocular nerves. They test the same neural pathways.

Pathway: Afferent receptors in the inner ear (semicircular canals and utriculus)

The stimulus can be:

- Mechanical (Rotation of the head in the horizontal plane) or
- Thermal (Ice cold water into the external auditory canal with head up 30° from horizontal)

Central: Afferent signals via the eighth cranial nerve to the brainstem. The connections between the third and sixth nerve nuclei occur via the *medial longitudinal fasciculus*



Figs. 1A and B: Examination of eye movements. The examination of eye movements is performed when the patient is conscious and can follow commands. In patients who are unconscious, a full range of eye movements cannot be tested. Only horizontal movements may be tested if there are horizontal roving movements. Explain to the patient and ensure the patient is comfortable. Ask the patient to look straight, and keep your hand on the head to prevent head movements. Instruct the patient to follow your finger (torch light can also be used instead of a finger). Move the finger in all directions and note down the eyeball movements. Figures A and B show the restriction of left eye lateral movement in this patient.

(MLF) embedded in the ARAS. The response of conjugate movement of the eyes thus tests the integrity of the *ARAS* (in which the MLF is embedded).

Efferent is via the third and sixth cranial nerves.

Technique:
Oculocephalic reflex ("dolling"): This test will also aggravate a pre-existing cervical spinal injury and should *not be done in case of any suspected injury to the cervical spine.* It may be necessary to disconnect the patient from the ventilator for 20–30 s (after preoxygenation with 100% oxygen and on a pulse oximeter) to be able to rotate the head adequately.

The caloric stimulation provides a greater stimulus than head rotation as the stimulus is more sustained.

If the test can be done safely,

Hold the patient's eyes open. The head is held in the neutral starting position and is turned briskly to one side. It is held in this position for 3–4 s and is then turned 180° briskly to the opposite side. The head is turned approximately once per second and remains in the new position for 3–4 seconds. Eye positions and movements are observed in each steady position.

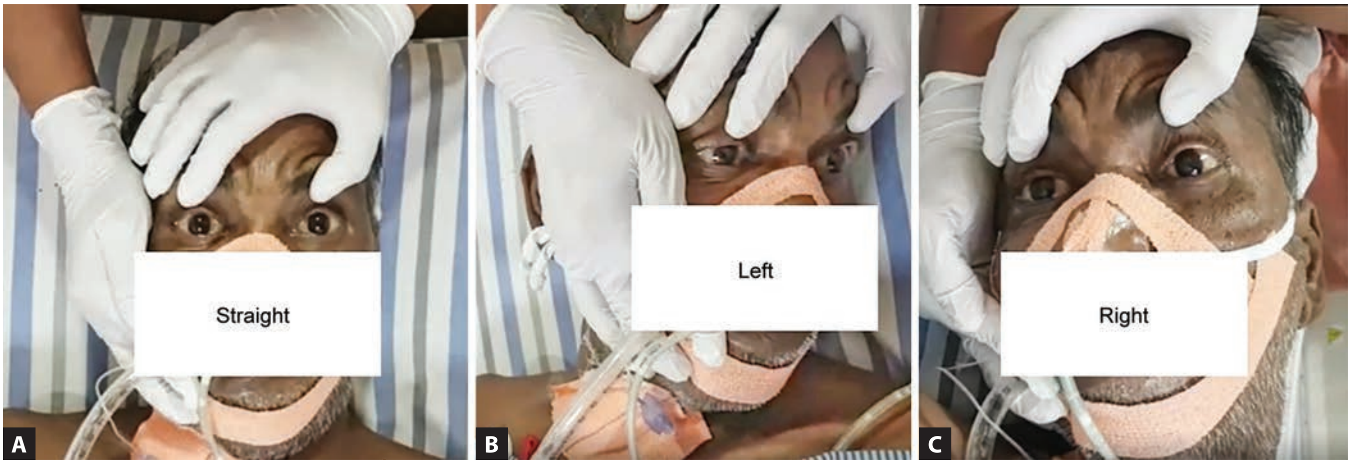
Positive doll's eyes reflex: Reflex conjugate deviation of the eyeballs in the direction *opposite to turning the head* ***(Figs. 2A to C)****.*

In an *alert individual*, the eyes will, within a fraction of a second, orient with the head (the oculocephalic reflex is suppressed in order for the eyes to see where the head is pointing). *A negative oculocephalic reflex occurs in a person who is awake because cerebral cortical activity suppresses it so that the eyes look where the head is turned.*

In a *comatose patient with damaged cerebral hemispheres* (*causing the coma*) but with a *functioning brainstem*, release phenomena can be seen. Passive rotation of the head will cause a conjugate deviation of the eyes to the opposite side. The eyes do not stay fixed (they move as in some dolls). The eyes are "out of phase" with the head. The reflex is *positive*.

In a *brainstem injury causing the coma*, this *reflex is lost* (*not suppressed* as in an alert person) and the head and eyes will move together (as if painted on). The reflex is *negative*.

Interpretation of response:
- *Positive:*
  - *Preserved brainstem:* Movement of eyes to opposite side. If this occurs, the brainstem is functioning, and the coma is cortical.
- *Negative:*
  - *Brainstem damage:* No movement—eyes move with the head as if they were painted eyes.
  - *Patient feigning coma* due to malingering or psychiatric illness (intact cortex): The cerebral



Figs. 2A to C: Doll's eye. Hold the head and neck by placing a hand over the back of the neck. Hold the endotracheal tube with another hand. The second operator will open both eyes and observer for eye movements. (A) Step 1—keep the head and neck in a neutral position (Rule out contraindications for the testing, e.g., unstable cervical spine); (B) Step 2—rotate the neck toward one side 90°. Observe for eye movements and patient for any discomfort; (C) Step 3—rotate 180° to the opposite side rapidly and observe for eye movements.

cortex suppresses contralateral eye movement (so that eyes can see where head is turning), eyes turn with head (as in brain stem dysfunction). However, other brain stem reflexes will reveal the true picture.

Thermal oculocephalic reflex (caloric test)

- A thermal stimulus is used for this test. An otoscopic examination should be done prior to testing to view the external auditory canal and ensure that the eardrum is visible. If there is wax (cerumen) in the external canal, it must be gently cleared. If there is a chronically perforated tympanic membrane, cold air (not water) may be used as a stimulus. *The presence of a ruptured eardrum does not invalidate the test.* Fractures to the base of the skull or petrous temporal bone may obliterate the response on that side.
- The patient's head is placed at 30° elevation from the supine position ensuring that the horizontal semicircular canal is horizontal (thus maximizing the stimulus). A soft catheter is introduced into the external auditory canal (a scalp vein needle with the butterfly flaps and metal needle cut off is a useful device). A volume of about 50 mL of iced water (up to 200 mL can be given) is given by slow injection into the external auditory canal with the eyes held open by an assistant. The eyes should be observed for at least a minute after the injection is completed. A period of 5 minutes should be allowed between tests before repeating the test on the opposite side to allow re-equilibration. Cold water (≤30°C) *mimics a head turn to the contralateral side.*
- With cold stimulation, the *normal response with intact brainstem and cerebral cortex* is that both the eyes turn toward the ipsilateral ear *(tonic conjugate deviation to ipsilateral ear), with compensatory horizontal nystagmus* (corrective component by the cortex) to the contralateral ear.
- In comatose patients *with an intact brainstem* (implying that the coma is due to bilateral cerebral cortical dysfunction), tonic deviation of the eyes occurs to the irrigated side *(tonic conjugate deviation of eyes to ipsilateral ear)*. However, as both the cerebral cortices are dysfunctional (causing the coma) the fast, corrective component (nystagmus) controlled by the cerebral cortex disappears.
- In a *comatose patient with brain stem dysfunction*, there is *no eye movement*.

  During testing for brain death, *any movement of either eye* (whether conjugate or not) *excludes* brain death.

  Warm water (≥44°C) *mimics a head turn to the ipsilateral side*. If it is used as the stimulus, the response will be the opposite of the above.

The acronym "COWS" (Cold opposite, Warm same) or the phrase "cold repels while warm attracts" summarizes the direction of the nystagmus (conventionally named as per fast component direction) not the tonic deviation.

In a *conscious patient*, eye movements can be deliberately tested as for a *standard neurological examination* **(Figs. 1A and B)**.

The following points described further are useful:

Gaze palsies: This may be due to weakness of muscles in one eye or due to a defect in the conjugate movement.

Monocular gaze palsies:

- The muscle affected in monocular gaze palsies can be picked up by requesting the patient to carry out the cardinal eye movements.
- The abducent nerve innervates the ipsilateral lateral rectus and hence its lesion would cause a loss of abduction in the same eye (ipsilateral).
- The trochlear nerve controls the superior oblique on the opposite side and hence it causes difficulty in looking down with the eye in adducted position (difficulty walking down the stairs due to diplopia). This is in the contralateral eye.
- All other eye movements are controlled by the oculomotor nerve of the same (ipsilateral) side.

Conjugate gaze palsies:

- *Horizontal gaze palsy:*
  - The frontal gaze center forces the eyes to look to the opposite side. Hence, an irritative frontal focus (seizure) will push the eyes to gaze away from the side of the lesion while a destructive lesion will prevent a conjugate movement to the contralateral side (the eyes at rest will look at the side of the lesion; **Fig. 3**).
  - The pontine gaze center does the opposite. It forces the eyes to look to the same side and hence a destructive pontine gaze center lesion will prevent conjugate movement to same side—so the eyes will look away from the lesion. An irritative lesion will have the opposite effect.
  - *Internuclear ophthalmoplegia:*
    - This is caused by a lesion in the MLF. The MLF contains fibers that connect the abducens nucleus to the contralateral oculomotor nucleus to perform horizontal conjugate lateral gaze.
    - It is not a peripheral nerve lesion as the MLF is in the brainstem. Unilateral lesions in the MLF disconnect the two nuclei. When the patient is asked to look laterally, the abducting eye is able to move but the *adducting eye ipsilateral to the abnormal MLF is unable to move medially. Nystagmus is often present in the abducting eye*. This symptom complex, which may be unilateral or bilateral, is termed *internuclear ophthalmoplegia (INO).*
    - *It is distinguished from a lesion of the medial rectus because the patient retains the ability to converge.*
- *Vertical gaze palsy:*
  - Vertical gaze becomes more limited with aging. It usually results from the midbrain lesions. There may be associated pupillary dilatation and vertical nystagmus with upward gaze.
  - *Parinaud syndrome* has a conjugate upward vertical gaze palsy resulting from a pineal tumor that compresses the midbrain or, less commonly, a tumor or infarct of the midbrain pretectum. It is characterized by the following:
    - Impaired upward gaze
    - *Collier sign:* Lid retraction
    - *Setting-sun sign:* Downward gaze preference
    - Dilated pupils that respond poorly to light but respond to accommodation
- *Downward gaze palsy:* Impaired voluntary downward gaze along with conjugate vertical gaze impairment along with preservation of reflex vertical movements is usually due to progressive supranuclear palsy.

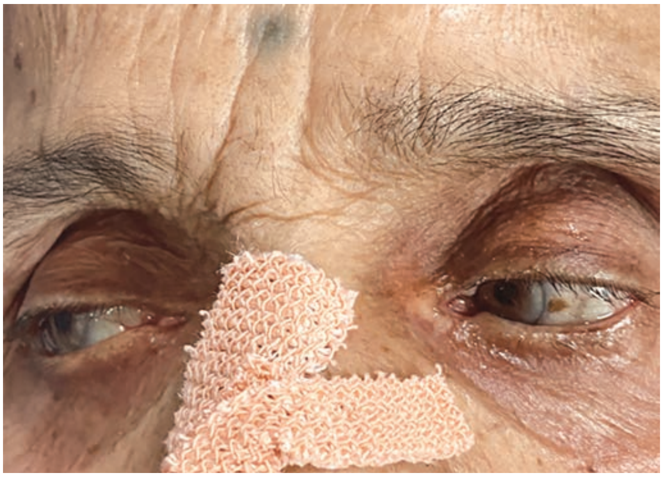


Fig. 3: The eyes deviate to the same side of the brain lesion. A computed tomography (CT) scan of the brain showed that the infarct is on the right side and eyes are deviated toward the right side.

Pupils

Always compare the two sides.

- Shape
- Size

- Reaction:
  - *Neuromuscular blockers do not cause changes in pupil size or reactivity.*
  - *Toxic-metabolic causes of coma can change pupillary size but reactions are maintained.*

Shape: Differences between the two sides can occur as a result of ocular surgery, trauma to the eyes, or due to inflammation (iritis), which causes adhesions between the pupil and anterior capsule of the lens.

Size: Anisocoria (unequal pupil sizes) can occur as a result of traumatic injury, increased intracranial pressure (ICP), nerve palsies, medication, or poisoning. The pupil is innervated by sympathetic fibers (via cervical sympathetic chain), which dilate it (mydriasis), and parasympathetic fibers (via the third cranial nerve), which constrict it (miosis). The size depends on the balance between these opposing forces. Mydriasis occurs due to sympathetic stimulation or parasympathetic blockage and miosis due to parasympathetic stimulation or sympathetic blockage **(Fig. 4)**.

Causes of *miosis* in the ICU: Pontine lesions, Horner syndrome, organophosphorus poisoning, and opioid administration

Causes of *mydriasis* in the ICU: Oculomotor lesions (brain stem injury, uncal herniation, and posterior communicating artery aneurysm), adrenergic stimulation as a result of medications (adrenaline or high-dose dopamine and nebulized salbutamol getting into the eyes), poisoning (amphetamine, parasympathetic block due to atropine, and tricyclic antidepressant overdose), or brain death.

Ambient light and anisocoria: The defective pupil's reaction to light is always less brisk than the normal pupil's reaction. If the pupillary asymmetry increases in bright ambient light, the larger pupil usually demonstrates a defect of constriction (has a mydriasis: parasympathetic dysfunction), whereas an increase of anisocoria in dim ambient light usually indicates a deficiency of dilation of the smaller pupil (has a miosis: sympathetic dysfunction). Thus, *mild mydriasis is best recognized in a well-lit environment* **(Fig. 3)**. A bright environment tends to constrict both pupils (direct and consensual reflexes), but the normal pupil will constrict more, accentuating the asymmetry. *Mild miosis is conversely picked up in a dim environment.*

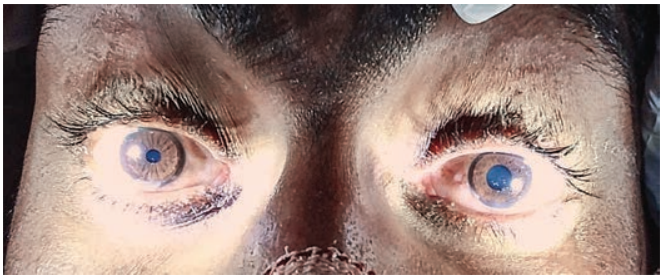


Fig. 4: Size of pupils and pupillary response to light. Note the left pupil is dilated.

TABLE 2: Pupil size, response to light in relation to the stage of the oculomotor nerve.

	Side of compression	***Opposite side***
Stage 1	Miosis	Normal
Stage 2	Mydriasis, impaired light reflex	Miosis
Stage 3	Mydriasis	Mydriasis

The nerve fibers of the oculomotor nerve pass through the interpeduncular fossa before passing between the posterior cerebral artery and the superior cerebellar artery to reach the cavernous sinus. During this course, the oculomotor nerve lies lateral to the posterior communicating artery. It then passes through the cavernous sinus and proceeds through the supraorbital fissure to reach the orbit of the eye. The *somatic (voluntary)* nerve fibers are bundled *deep inside* the nerve, whereas the *autonomic fibers* to the pupil are *superficial* on the outside of the nerve. Knowing the spatial layout of these fibers will help one understand the various forms of presentation in third nerve palsies.

In a patient who has increased ICP as the brain is pushed down, the third nerve is compressed leading to *Hutchinson's pupillary changes* **(Table 2)**.

As the parasympathetic fibers are superficially located, a compressive pathological process affects pupillary size before ocular movements are affected. Compression of the oculomotor nerve can also occur due to the following:

- An aneurysm (in posterior cerebral, superior cerebellar, posterior communicating)
- Intracranial space-occupying lesion causing uncal herniation

In contrast, in microvascular vascular lesions (diabetes mellitus, hypertension, and vasculitis), the pupillomotor fibers are relatively spared from the ischemic injury. This is because the core of the extra-axial segment of the oculomotor *nerve is infarcted while the superficial fibers are spared. The earliest manifestation may be only ptosis, which can then progress to involve other extra-ocular muscles innervated by the oculomotor nerve.*

Hippus*:* Spontaneous changes in the pupillary size may be observed and are termed *hippus*. It can occur during recovery from a third nerve palsy or more rarely, it may be a sign of seizure activity in a patient who is paralyzed with neuromuscular blocking agents, and therefore the motor manifestation of the seizure activity is blocked.

Reaction: This may be due to light or accommodation.

Light reflex: Initially, shine the light from the lateral side (avoid shining from anterior to prevent triggering the accommodation response; if the patient is conscious and focuses on the light, the accommodation response will be activated). If no response is seen, the light can be shone directly. Both pupils need to be observed for any reaction. The response of the one into which the light is shown is the *direct response* and the reaction of the other pupil is the *consensual response*. Next, move the torch in an arc from pupil to pupil. If the eye has any optic nerve dysfunction, it will show a paradoxical dilatation when the torch is moved from the normal to the affected eye. This is the *afferent pupillary defect* (Marcus Gunn pupillary sign; **Table 3**). This occurs because the afferent impulse from the affected eye is reduced, so much so that when the light is shifted from the normal to the abnormal eyes, a paradoxical dilatation is noted. The pupillary dilatation *after a consensual constriction* (when the light is first shone in the normal eye and then moved to the abnormal side) dominates over the weak stimulus when light is shown in the affected eye (due to optic nerve disease).

The accommodation response: This is done by requesting the patient to look afar and then to a finger at about 30 cm midway between the eyes. The accommodation response involves constriction of both pupils **(Table 4)**. It involves pathways from the visual association cortex to the third nerve nucleus. In conjunction with the light reflex, it can give useful information.

It cannot be done in unconscious patients.

TABLE 3: Interpretation of pupillary light reflex.

Interpretation of pupillary light reflexes

		Direct light reflex (eye into which light is shone)	
		Absent	*Present*
Consensual light reflex (opposite eye)	*Absent*	Ipsilateral optic nerve lesion	Contralateral oculomotor nerve lesion
	Present	Ipsilateral oculomotor lesion	Normal

TABLE 4: Interpretation of accommodation response of pupillary light reflex.

Interpretation of pupillary reflexes

		Pupillary light reflex	
		Absent	*Present*
Accommodation response	*Absent*	Bilateral optic nerve lesion, Parinaud syndrome (some)	Occipital cortical blindness, midbrain lesion (rare)
	Present	Ciliary ganglion lesion, Holmes-Adie, Argyll-Robertson, Parinaud syndrome (most)	Normal

Trigeminal Nerve: V

The trigeminal nerve has three testable components as follows:

1. Motor
2. Sensory
3. Reflexes:
    i. Corneal
    ii. Jaw jerk

The motor component supplies the jaw muscles: Closing (masseter, temporalis) and opening (pterygoids). A lesion on one side causes the jaw to deviate to the ipsilateral side (side of the lesion) upon opening the mouth.

The sensory component carries the sensation of touch, pain, temperature, and proprioception from the face. Only touch and pain are tested.

Testing reflexes related to the V nerve are more useful in the ICU than testing the other components.

Corneal Reflex

The afferent is via the V sensory and efferent via the facial nerve (VII).

Stimulus: Remove any contact lens. Ask the patient to look away (so that the examiner's action is not visible) and

TABLE 5: Interpretation of corneal reflex.

Interpretation of corneal reflexes

		Direct corneal reflex	
		Absent	*Present*
Consensual corneal reflex	*Absent*	Ipsilateral trigeminal nerve lesion	Contralateral facial nerve lesion
	Present	Ipsilateral facial nerve lesion	Normal

touch the lateral aspect of the cornea with a wisp of cotton. Touch the cornea not the conjunctiva. Touch only the outer part of the cornea so that the response is not in response to a visual stimulus (menace response) and also to prevent any possibility of a corneal abrasion in the pathway of light reaching the lens.

Response: Closure of both eyelids. The *direct corneal reflex* refers to closure of the eyelid on the ipsilateral side and the *consensual corneal reflex* refers to closure of the eyelid on the contralateral side.

The response can be interpreted as shown in **Table 5**.

Jaw Jerk (Masseter Reflex)

The patient's mouth is left open slightly. The examiner's thumb is placed on the lower jaw in the mental region above the chin and the other four fingers over the masseter muscle. A tap on the examiner's thumb with a tendon hammer normally elicits either no response or a slight contraction of masseters (palpable by the fingers on the muscle) or resulting in mouth closure (observation). Its significance is only when it is exaggerated—indicating a bilateral upper motor neuron lesion above trigeminal motor nucleus (above pons). This is commonly seen in pseudobulbar palsy.

Facial Nerve: VII

The facial nerve has the following components for testing:

1. Motor: *Facial movements*—wrinkling forehead, closure of eyes, and lip and cheek movements (blowing up cheeks, smiling, and showing teeth)
2. Sensory: *Taste* from the anterior two-thirds of tongue
3. Reflexes:
   a. Corneal reflex
   b. Glabellar tap

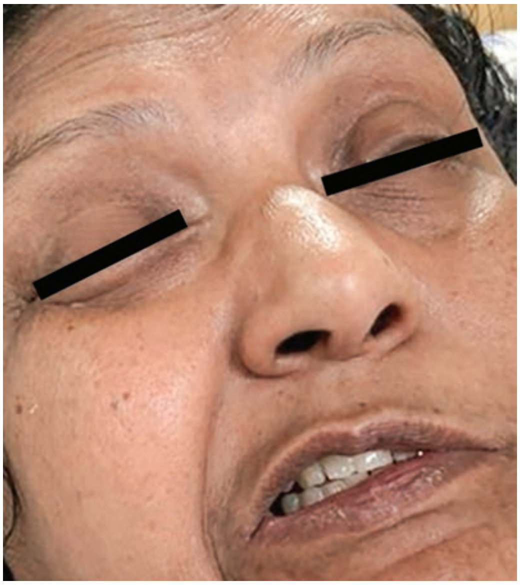


Fig. 5: The deviation of angle of mouth to the right side and incomplete closure of left eye with preserved wrinkling of forehead on both sides indicates upper motor neuron (UMN) facial nerve involvement of left side.

Motor

The motor component innervates the forehead (wrinkling) orbicularis oculi (closure of eyes) and the muscles of facial expression. First, observe facial asymmetry. Mild asymmetry can be normal. In a conscious patient, testing involves asking the patient to wrinkle their forehead, shut their eyes tightly, blow up cheeks, smile, and show teeth with teeth in apposition **(Fig. 5)**. In an unconscious patient, supraorbital pressure can reveal the weakness of facial muscles.

The facial nerve nucleus supplies the ipsilateral facial muscles via the LMN.

The UMN supply from the cortex to the facial nerve nucleus has a unique pattern: the upper part of the facial nerve nucleus is supplied from both cerebral cortices whereas the lower part of the facial nerve nucleus receives only UMNs from the contralateral cortex. This transition from bilateral control to unilateral control is gradual so that in some individuals, the top third of the nucleus (controlling wrinkling of the forehead) has bilateral supply whereas in others it is the top half (controlling forehead wrinkling and eye closure) that has the bilateral control. It is important to keep this anatomy in mind because in a UMN facial nerve lesion, only the lower facial movements (contralateral to the UMN lesion) are affected (the upper part can be controlled through the ipsilateral cortex), whereas in an LMN facial lesion, one half of the face (ipsilateral, both upper and lower part) is affected.

It is also important to note that in a facial palsy due to a cortical lesion, emotional facial movements (crying or smiling) may be spared as they originate in other areas.

Testing the sensory component of taste is not useful in the ICU setting.

Reflexes

Corneal reflex has already been described in detail in the section on the examination of cranial nerves under trigeminal nerve.

Glabellar tap: The *glabellar reflex*, is also known as the "*glabellar tap sign.*" It is elicited by repetitive tapping of the glabella. The afferent signal is transmitted by the trigeminal nerve to the brainstem and the efferent signals go via the facial nerve to the orbicularis oculi muscle, causing blinking. The normal response to the first several taps is by blinking but if tapping is done persistently, it would normally lead to suppression of blinking. If the blinking continues with persistent tapping, it is a frontal lobe release sign, known as *Myerson's sign*. It can be present in those who have Parkinson disease, frontal lobe disease, and pseudobulbar palsy.

Vestibuloauditory: VIII

The functions of hearing and balance are subserved by the VIII cranial nerve.

A detailed testing of the auditory component is not useful in the ICU. However, bilateral deafness makes it difficult to waken a patient by calling her/him and can obviously make other testing difficult.

Vestibular function testing is useful in an unconscious patient (see the section on the examination of cranial nerves under ocular Nerves under oculocephalic reflex).

Glossopharyngeal-Vagus: IX, X

These are usually tested together.

IX: Transmits touch sensation from the nasopharynx and pharynx and taste from the posterior third of the tongue.

X: Sensory fibers from the pharynx and larynx; motor fibers to the pharynx and larynx **(Fig. 6)**.

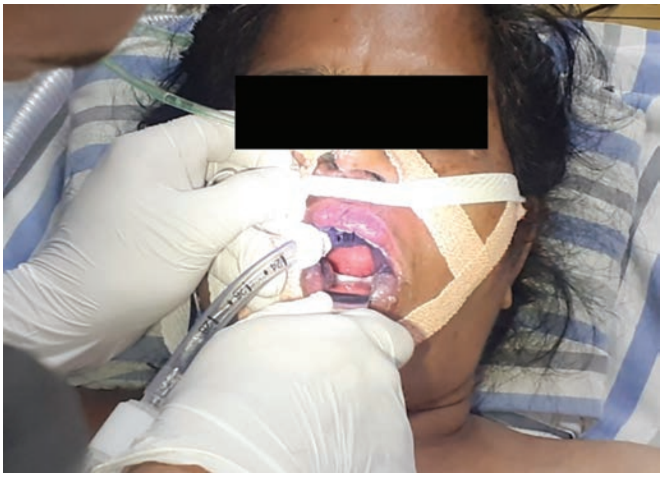

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Fig. 6: Examination of a patient for gag reflex and uvula movement. First, explain the procedure to the patient. Keep the patient in a comfortable position, preferably upright. Tell the patient to open the mouth, and gently introduce the tongue depressor to depress the tongue enough to visualize the posterior pharynx and uvula. If the patient is cooperative, tell the patient to say "ah", and look for uvula movement. A swab stick can be used to gently touch the posterior pharynx to check the gag reflex.

It is of significance in two situations:

1. Absence of gag reflex makes nasal regurgitation and pulmonary aspiration likely
2. Bilateral laryngeal abductor muscle paralysis can result in life-threatening stridor. This can occur post-extubation and will need re-intubation followed by tracheostomy.

Accessory: XI

This is a motor nerve and its testing is not useful in the ICU setting. It has central and spinal components—the central component provides fibers to the vagus and the spinal component (C1 to C5) innervates the trapezius and sternocleidomastoid muscles of the ipsilateral side.

Hypoglossal: XII

Testing this nerve is not useful in the ICU. It is interesting to note that it is tested by requesting the patient to protrude the tongue—the tongue will deviate to the weak side because the intact genioglossus pulls the tongue forward contralaterally without any resistance from the weak side (which, if active, would have kept the protrusion in the midline; **Fig. 7**).

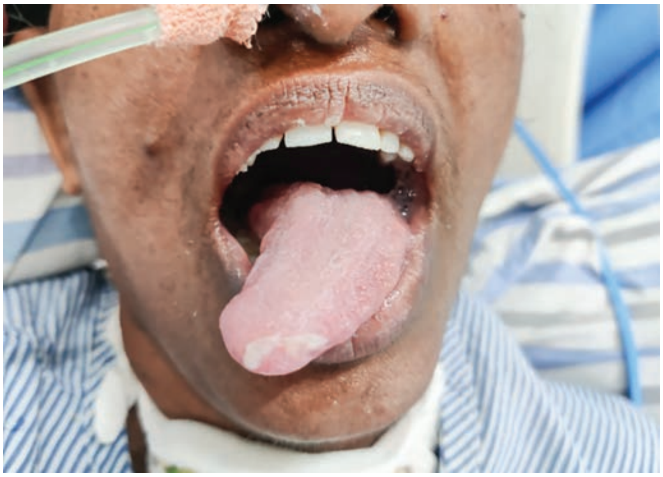

Fig. 7: Assessment of XII cranial nerve. The tip of the tongue has deviated toward the right side (weak side) due to the action of an intact nerve on the left side.

BEDSIDE EVALUATION WITH DEVICES

The traditional clinical skills of inspection, palpation, percussion, and auscultation (with the historical modification by orthopedicians of adding movement and measurement with deletion of percussion and auscultation) can be augmented by using bedside devices.

Imaging the brain has deepened our understanding of the pathophysiology of clinical signs. In a critically ill patient, this includes angiogram, CT scan, magnetic resonance imaging (MRI), isotope imaging, etc., all of which usually need patient transfer to the appropriate location.

However, there are bedside devices that can complement clinical examination, including the following:

1. Ophthalmoscopy
2. Ultrasound
3. Electroencephalogram (EEG)
4. Nerve conduction and electromyography (EMG)
5. Bedside CT and MRI

This discussion will focus on 1 and 2, which can be done by an intensivist.

Ophthalmoscopic Evaluation

The ophthalmoscope can be used to detect local eye disorders and systemic problems that can be reflected in the eye. In the context of a neurological evaluation, it can also be used to magnify and observe minimal pupillary reactions and mild nystagmus.

TABLE 6: Differences between papilledema and papillitis.

Papilledema	***Papillitis***
Usually bilateral	Usually unilateral
Visual acuity preserved, enlarged blind spot	Early loss of visual acuity
No ocular pain but headache may be present	Pain on eye movement
Spontaneous venous pulsations absent	Spontaneous venous pulsations present
Splinter hemorrhages may be present	No splinter hemorrhages
Normal pupillary reaction	Marcus Gunn pupils

This discussion will focus on two acute/subacute conditions:

1. Increased ICP
2. Bleeds in the eye

Increased Intracranial Pressure

Spontaneous retinal venous pulsations (seen using an ophthalmoscope) are present with normal ICP. However, it may not be visible in about 20% of normal individuals, so its absence does not necessarily indicate a high ICP. They may be elicited by exerting mild eyeball pressure in a person in whom it is not seen, but this could give a false positive sign that ICP is normal and hence should not be done.

Papilledema implies a swollen optic disc due to raised ICP. If it is due to a raised ICP, it is usually bilateral. Blurring and loss of a distinct disc margin are two of the earliest signs. Splinter hemorrhages around the disc can also be associated with papilledema.

Many conditions can imitate papilledema (pseudopapilledema). These include hypermetropia (no true disc protrusion), medullated nerve fibers (usually unilateral), and colloid bodies of the disc.

Nonpapilledematous optic disc swelling (papillitis) may be due to infection, inflammation, or demyelination (optic neuritis, multiple sclerosis, central retinal vein or artery occlusion, diabetic papillopathy, malignant hypertension or due to toxins).

The differences shown in **Table 6** may be useful to note.

Bleeds in the Eye

Splinter and flame-shaped: Blood follows the path of least resistance, which is along and between the nerve fibers in the retina and not across them. In the inner nerve fiber layer, the axes of nerves are perpendicular to the light being shone—hence hemorrhage in this layer is feathery. Splinter-shaped hemorrhages are bleeds in the inner nerve layer. The individual splinter is a line of blood pooling between nerve fibers perpendicular to the light from the ophthalmoscope. A larger collection will have a flame-shaped or feathery edge—a flame-shaped hemorrhage is a fusion of splinter hemorrhages.

Subhyaloid hemorrhage (preretinal): Subhyaloid hemorrhages can be seen most often after a rupture of an intracranial aneurysms or sometimes after head injury. The shape of preretinal hemorrhages vary depending on patient position—round top when seen with the patient supine (as in an unconscious patient) or flat at the top (if patient is erect). They always obscure the retinal vessels.

Ultrasound

Optic Nerve Sheath Ultrasound

Optic nerve sheath ultrasound (ONSUS) is a bedside procedure that can be used to evaluate for increased ICP.

The optic nerve is an extension of the brain. The intra-orbital component is encapsulated by dura, arachnoid, and pia matter. The meningeal cover of the optic nerve is a continuation of the dural and subarachnoid space. This allows the optic nerve sheath to transmit cerebral spinal fluid pressure and its changes. The bulbous portion of the optic nerve, approximately 3 mm posterior to the globe, appears to be the most distensible and sensitive to changes in ICP.

Optic nerve sheath diameter (ONSD) is a reliable marker of intracranial hypertension. ONSD changes with ICP in real time. Monitoring it is useful in ICUs (where invasive ICP monitoring is not routinely available) in order to recognize early intracranial hypertension and for recognizing possible brain death.

The probe used is one with the highest available frequencies (12–14 MHz linear array transducer) because of the relatively shallow depth, small volume of the region of interest (ROI), and very low attenuation in ocular media. Higher frequencies offer better resolution and smaller, easier-to-handle probes with a small contact surface.

The upper limit of the normal ONSD includes:

- 4 mm in infants
- 5 mm in children and adults
- ONSD shows linear increment till 7.5 mm, after which it plateaus off.

Systematic reviews and meta-analyses have shown that ONSD of >5.70 mm has a concurrent ICP value >20 mm Hg.

Transcranial Doppler

Intracranial neurovascular examination is done by using a transcranial Doppler (TCD) study. TCD is a painless, noninvasive, radiation free, and easily repeatable bedside test. It measures the rate and direction of blood flow inside intracranial vessels (arteries in the circle of Willis). The ultrasonic beam that is emitted from the TCD probe crosses the skull at the target location and is reflected from the erythrocytes, which flow at different speeds in the cerebral vessels. The reflected signals are recorded. It provides an indirect assessment of the cerebral vessels' diameter. An ultrasound probe of low frequency is used (2 MHz) because soft tissues and bone attenuation are low at this range compared with higher frequencies.

It provides information regarding cerebral blood flow velocity (CBFV). The basic principle of TCD ultrasonography is based on the fact that the CBFV in a cerebral artery is inversely proportional to the diameter of that artery.

Three *acoustic windows* to evaluate the cerebral arteries are available:

1. Transtemporal window (between the eye and ear pinna) to assess the middle cerebral artery
2. Transorbital window to assess the ophthalmic artery
3. Transforaminal window (across the foramen magnum) to evaluate the vertebral arteries

The use of TCD helps in the following:

- Early detection of cerebral vasospasm following subarachnoid hemorrhage (SAH)
- Detection of ischemic stroke and monitoring of efficacy of treatment in patients with hyperacute stroke
- Early diagnosis of hyperperfusion syndrome after carotid endarterectomy
- Monitoring of arteriovenous malformation (AVM) after definitive treatment
- Confirmation of brain death

Its limitations are as follows:

- High operator dependency
- Measurements of velocity may be affected by multiple factors such as age, gender, hematocrit value, differences in the partial pressure of CO_2 in blood, and thickness of the skull bone

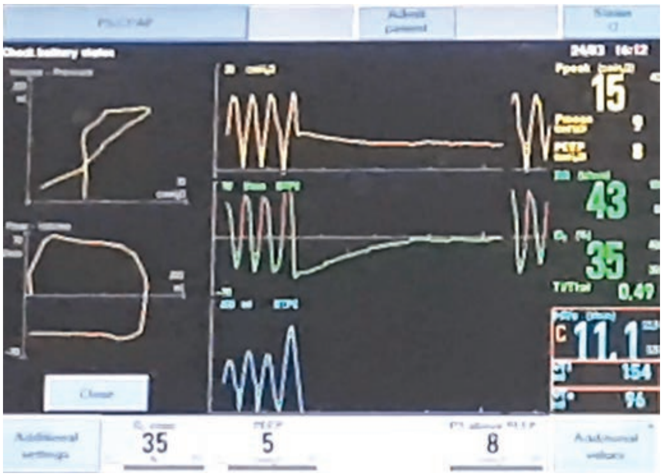

Fig. 8: Biot breathing: It is characterized by regular deep respirations interspersed with periods of apnea. This is the ventilator graphics from a patient who had a pontine infarct.

- Measurements are also limited to the large basal arteries and it can only provide an index of global and not local cerebral blood flow velocity
- Superficial AVM and those located in the parietal, occipital, and cerebellar regions are difficult to detect.

SUMMARY

The examination of the central nervous system in a critically ill patient comprises of examination of the brain, cranial nerves, peripheral nerves and muscles systematically. Examination of the brain and cranial nerves is included in this section and the rest in the next section. The examination is done within the constraints relevant to a critically ill patient with the goal of assessing the location and extent of neurological dysfunction.

The brain is evaluated in terms of the level and content of consciousness. The twelve cranial nerves are then examined systematically.

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CASE SCENARIO 2

A 20-year-old girl from a village was brought to the accident and emergency department of a tertiary care center with a history of being unarousable after going to sleep on the verandah of her house in the village. There was no history of ingestion of any poison.

The patient was deeply cyanosed. She was promptly intubated and put on mechanical ventilation. On detailed examination, she had a Glasgow Coma Score of 2T/10 with total ophthalmoplegia, dilated pupils, and absent pupillary, vestibulo-ocular, and corneal reflexes. There were no facial movements or limb movements in response to pain. Deep tendon reflexes were not elicitable. Abdominal and plantar responses were absent.

The heart rate was 100 beats/min, blood pressure 100/60 mm Hg, and respiratory rate was <10 breaths/min with an SpO_2 of 70%. Temperature was 36.6°C. Cardiorespiratory examination was normal. Abdominal examination showed possible fang marks on the right lateral abdomen with no local signs of inflammation.

The patient had been completely paralyzed on arrival and had thus been apneic for an indeterminate period of time. Therefore, hypoxic encephalopathy leading to brain death was suspected. However, in view of her age, potential reversibility and presence of fang marks, it was decided to treat her for 72 hours and reassess.

Thinking Through

This patient was unconscious and hypoxic on arrival.

The first question is whether the patient needs cardiopulmonary resuscitation (CPR).

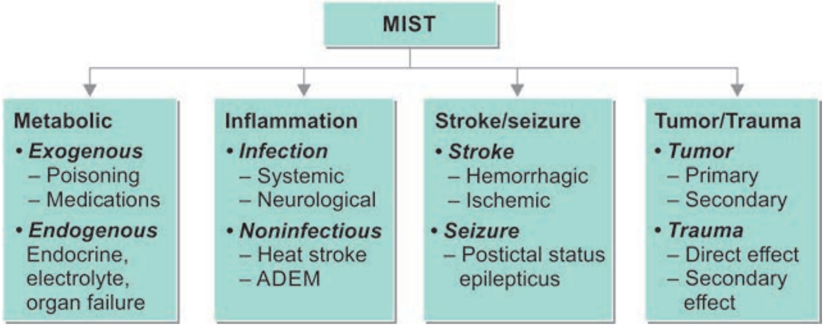
However, there is a pulse and blood pressure but respiratory effort was inadequate, and because diagnosis and treatment must proceed in parallel, she is intubated.

She had areflexic quadriparesis with nonreacting pupils.

The next question is: What is the cause of neuromuscular paralysis and pupillary unresponsiveness?

A rapid mnemonic to remember the causes of coma in terms of pathophysiology is MIST as depicted in **Flowchart 1**.

Flowchart 1: Mnemonic to remember the causes of coma in terms of pathophysiology.


MIST
Metabolic
• Exogenous
– Poisoning
– Medications
• Endogenous
Endocrine, electrolyte, organ failure
Inflammation
• Infection
– Systemic
– Neurological
• Noninfectious
– Heat stroke
– ADEM
Stroke/seizure
• Stroke
– Hemorrhagic
– Ischemic
• Seizure
– Postictal status epilepticus
Tumor/Trauma
• Tumor
– Primary
– Secondary
• Trauma
– Direct effect
– Secondary effect

(ADEM: *acute disseminated encephalomyelitis)*

Rapid testing for glucose is easily done using finger-prick sample.

The last two boxes (stroke/seizure, tumor trauma) are usually made out from a combination of history and localizing signs.

In the present scenario (lack of positive history and no localizing signs), the first two boxes are more likely as a cause for the coma.

In view of the acuity of onset, the *first box is* most likely and the highest priority would be poisoning as endogenous metabolic causes are usually slower (except for hypoglycemia).

In addition, the history in a person who has taken poison is notoriously unreliable so poisoning is still a possibility.

The one point against poison ingestion is the unresponsive pupils.

The additional finding of possible fang marks makes the diagnosis of envenomation likely. However, krait bites are notoriously difficult to detect due to lack of local signs.

In view of the potential reversibility of envenomation, she is given further treatment.

Subsequent Events

She was shifted to the ICU. Supportive therapy was initiated. In view of possible snake bite, she was given polyvalent antivenin but with no immediate change in neuromuscular status. Hematological and biochemical values were normal. A baseline cold caloric stimulation test was performed and was negative. CT brain was normal and an MRI subsequently showed no gross abnormality. Patient remained in a comatose state for the next 2 days and showed no change in neurological status. On day 3 of admission, the patient started to cough on endotracheal suctioning (this had earlier been absent). Further examination revealed slight pupillary constriction to bright light. Over the next 48 hours, the patient had a gradual improvement in ophthalmoplegia and muscle power of the extremities. Higher mental functions appeared to be intact. Mechanical ventilation was continued for another 5 days due to persistent respiratory weakness. After a week, the patient was successfully weaned off mechanical ventilation.

Chapter

9

Central Nervous System Examination in a Critically Ill Patient: Part 2 Neuromuscular System

Farhad N Kapadia, Jare Jagannath Uddhavrao

ABSTRACT

Central nervous system examination is integral part for the diagnosis and management of critically ill patients. An initial concise and focused neurological evaluation should be done on presentation to intensive care unit (ICU) or emergency room or if a new neurological complication has developed. Initial evaluation includes assessment of consciousness [usually by using the Glasgow Coma Scale (GCS)], pupillary size and reactivity to the light, motor response, plantar reflex, respiratory patterns, extraocular eye movement, and any other focal neurological deficit. It should be kept in mind that evaluation should be started with airway-breathing-circulation (ABC). As one does the examination, one tries to answer three questions. One, are the neurological changes due to CNS illness, or due to something outside the CNS, affecting its function. Two if neurological, where is the lesion, and three, what is the lesion.

INTRODUCTION

In an emergency, immediate evaluation of the vitals and the cardiorespiratory system takes precedence over the neurological examination. Once it is established that the person does not need immediate resuscitation, one evaluates the level of consciousness and then proceeds to a complete systemic examination including the neurological system. Throughout this evaluation, one must monitor for non-neurological complications such as airway obstruction, or impaired ventilation. These complications may evolve after the initial central nervous system (CNS) examination.

The classical neurological examination consists of evaluation of the higher functions, the cranial nerves (CN), the motor system, the sensory system, and the reflexes, usually in that order. This has to be modified to a more concise and focused examination for the intensive care unit (ICU) or emergency patient.

As one does the examination, one tries to answer three questions. One, are the neurological changes due to CNS illness, or due to something outside the CNS, affecting its function. Two if neurological, where is the lesion, and three, what is the lesion.

In today's world, rapid imaging often precedes a detailed clinical evaluation. If imaging precedes one's initial clinical examination, then obviously, the findings should be factored in when doing a further clinical examination.

The earlier preceding chapter on neurological examination deals with the patients with altered consciousness, confusional states, or altered cognition and behavior. In this chapter, we will concentrate on the patients with abnormal CNS findings with retained consciousness and comprehension. There will be some overlap with the preceding chapter.

CASE SCENARIOS: INTRODUCTION

These are all based on actual patients treated by the authors.

Case Scenario 1

A 65-year-old hypertensive and diabetic male patient was noted to appear confused by his family at 9:30 AM. *He seemed to have problems communicating with them. A little later they noticed some face asymmetry and right upper and lower limb weakness. He reached the hospital at 11:00* AM. *A rapid initial examination noted that he was fully consciousness but poorly communicative (clinically interpreted as aphasia). All the vital signs were normal as was the* SO_2 *and the glucose. The blood pressure was* *raised at 170/90 mm Hg. A rapid systemic examination was unremarkable. Neurological examination showed a right upper motor neuron (UMN) pattern facial nerve weakness and he had grade 3 power in the right upper and lower limbs. The right plantar was upgoing. The rest of the neurological examination was unremarkable. A diagnostic test was done at 11:15 AM and appropriate treatment was started at 11:30 AM.*

Case Scenario 2

An elderly male had COVID-19 and was treated at home. He did not require oxygen or steroids. The family noted decreasing mobility over the following 1–2 weeks. He was unable to mobilize even to the bathroom or for meals. On admission, he was referred to a neurologist who noted hypotonia, a generalized decrease of power (grade 4), and a bilateral facial palsy resulting in a "fish mouth" appearance. The tendon reflexes were absent. The basic investigations were unremarkable. A diagnostic test was done and appropriate treatment was started.

Case Scenario 3

A 70-year-old female with known asthma/chronic obstructive pulmonary disease (COPD) was admitted for an exacerbation that did not respond to usual home treatment including oral steroids. On admission, she was in respiratory distress and the arterial blood gas (ABG) analysis showed CO_2 *retention. She was given standard treatment but needed noninvasive ventilation (NIV) for increasing periods of time with persisting and then worsening dyspnea. A diagnostic tests was done on day 3 of her nonresolving illness.*

Neurological Examination of the Patient with Preserved Consciousness and Normal Higher Functions

The clinical history is usually more important that the physical examination in the context of new onset motor weakness with preserved consciousness in determining the diagnosis. A brief but focused history usually points to the diagnosis. The initial clinical examination and imaging are then used to confirm the diagnosis or to consider alternative diagnoses. Subsequently, a more detailed history and investigations further confirm or alter the initial diagnosis.

An initial concise and focused neurological evaluation should be done on presentation to ICU or emergency room. It should also be done if a new neurological complication has developed. This initial evaluation includes assessment of consciousness [usually by using the Glasgow Coma Scale (GCS)], pupillary size and reactivity to the light, motor response, plantar reflex, respiratory patterns, extraocular eye movement, and any other focal neurological deficit. It should be kept in mind that evaluation should be started with airway-breathing-circulation (ABC). It should always be associated with timely identification and treatment of common treatable causes such as glucose in hypoglycemia, antiepileptics in convulsion, naloxone in opioid overdose, or thiamine in alcohol intoxication.

Question 1: Is this part of a systemic illness with neurological manifestations?

The presence of abnormal pupils, asymmetrical neurological findings, or abnormal reflexes points to the problem being neurological. (One should clarify that the pupil changes are not due to prior surgery or injury, and other abnormal neurological findings are not due to a pre-existing neurological event such as a stroke). If feasible, resting eye movement should be checked, if there is no resting eye movement, doll's eye and extraocular movement should be checked and locked-in syndrome should be ruled out in which only vertical eye movement is preserved and clinically patient appears to be in coma, but is conscious. Fundoscopic examination may help to find out the cause of alteration neurological function such as papilledema, which usually indicates raised intracranial pressure. Evaluation for meningeal involvement with a stiff neck should also be done at this stage. Some patients with progressive neurological illness such as Parkinson disease may have a stiff neck. Here the stiffness will also be present in lateral rotation, whereas diseases such as meningitis and subarachnoid hemorrhage will only have stiffness to flexion.

Question 2: Where is the lesion?

A detailed CNS examination, discussed later, will allow the clinician to determine if the problem is in the cerebrum, the brainstem or cerebellum, the spinal cord, the peripheral nerves, the myoneural junction, or the muscles.

Table 1 shows the clinical manifestations at different levels of the neurological system.

By quickly going over these features during the initial examination, one can make an initial evaluation of the site of the lesion.

TABLE 1: Clinical manifestations at different levels of the neurological system.

Site of lesion	***Clinical sign***
Cerebrum	• Bilateral cerebral lesion causes altered mental status or cognitive impairment • Unilateral weakness and sensory abnormalities including head and limbs • Seizures • Visual field abnormalities • Movement disorder (e.g., diffuse incoordination, tremor, and chorea)
Brainstem	• Isolated cranial nerve involvement either single or multiple • Crossed hemiplegia means LMN involvement of cranial nerve on the side of lesion and hemiplegia or hemisensory loss on the opposite side of the body
Spinal cord	• Back pain may be radiating, local spinal tenderness • Weakness and sensory involvement sparing the head • Sensory level • Bowel and bladder involvement • LMN signs at the site of lesion and UMN signs below the site of the lesion
Spinal roots/ radicles	• Radiating pain to particular dermatome • Weakness and sensory involvement of area supplied by spinal root • Loss of reflexes
Peripheral nerve	• "Stocking and glove" distribution of sensory loss • Loss of reflexes • Weakness and sensory loss in nerve distribution
Neuromuscular junction	• Bilateral weakness causing ptosis, diplopia, dysphagia, and proximal limbs weakness • Weakness is more on exertion and improves on rest • Sensation remains normal
Muscle	• Bilateral proximal or distal weakness • Sensation remains normal

(LMN: lower motor neuron; UMN: upper motor neuron)

Question 3: What is the lesion?

Once the location of the lesion has been established, one can enumerate the differential diagnoses for that lesion and investigate it accordingly.

Sudden onset of neurological problem is usually due to vascular causes such as thrombosis, bleeding, embolism, trauma, infections, Todd's paralysis (postictal), or metabolic causes such as hypoglycemia, hypokalemia, and toxins.

Subacute onset of neurological problem is due to demyelinating diseases, chronic infection, slow growing tumors, and nutritional deficiency such as vitamin B_{12}.

Chronic onset of neurological problem usually indicates degenerative disease or tumors.

By quickly going over these features during the initial examination, the clinician can arrive at a working diagnosis of the site and the cause of the neurological problem. At this point, one should be certain that the patient is stable and the initial appropriate treatment has been administered.

Detailed Neurological Examination

One then proceeds to a more detailed neurological examination, including the CN, the motor system, the sensations, and the reflexes. This detailed examination is necessary to confirm the initial diagnosis and to find any additional features that may require management.

The Cranial Nerves

The examination of the CN is usually done in two separate contexts. One, in a patient with altered consciousness or higher functions, and two, in a patient with preserved consciousness and higher functions. The below discussion is focused on the latter patient. Inevitably, the presence of CN abnormalities points to a lesion in the brain or brainstem, and occasionally in the cerebellum, the meninges, the cranium, the cranial foramen, or in the nerves themselves. Rarely, like in Horner syndrome, the lesion may be extracranial. Disease of the myoneural junction such as myasthenia or myopathies may involve the CN:

- Pure motor CN are III, IV, VI, XI, and XII
- Pure sensory CN are I, II, and VIII
- Mixed motor and sensory CN are V, VII, IX, and X

Olfactory nerve: The function is to get the sense of smell. We do not examine the olfactory nerve often in critically ill patients. Common causes of loss of smell are upper respiratory tract infection including COVID-19. Unilateral loss of smell indicates olfactory bulb compression. Other rare causes include head trauma, frontal lobe tumor, or chronic basal meningitis.

Optic nerve: The function is to get the sense of vision, which is checked with visual acuity, field of vision, color vision, pupillary response, and ophthalmoscopic examination of the optic nerve head and fundus.

At the bedside, visual acuity is checked with finger counting at a distance of 1 m. Detailed examination of visual acuity requires equipment and patient's cooperation, which may not be feasible in the ICU environment. Often the patients are too sick to be tested.

Visual field is checked with confrontation method.

Monocular loss of vision is usually ophthalmic. Neurological causes of monocular blindness are optic neuritis, multiple sclerosis, ischemia-giant cell arteritis, retro-orbital tumor, retinal artery, and vein occlusion. Binocular vision loss occurs in stroke, raised intracranial pressure, methyl alcohol poisoning, and optic chiasma compression.

Fundoscopic examination may be done to look for the papilledema. Papilledema commonly occurs in raised intracranial pressure, cavernous sinus thrombosis, superior vena cava syndrome, hypertension, central retinal vein occlusion, Graves' disease, hypercapnia, subarachnoid hemorrhage, meningitis, and methanol and lead poisoning.

Figure 1 shows the various field defects depending on the site of the lesion.

Oculomotor, trochlear, and abducent CN: These three nerves should be examined simultaneously because these nerves act as unit and control extraocular movement.

Figure 2 shows the action of the oculomotor muscles. The following should be examined when evaluating these nerves:

- *Ptosis:* Ptosis means narrowing of palpebral fissures. It commonly occurs due to third nerve lesion and Horner syndrome.

  Causes of unilateral ptosis: Compression of the third nerve by the uncus of temporal lobe during cerebral herniation, compression of the third nerve by aneurysm of posterior communicating artery, posterior cerebral artery, or internal carotid artery, and cavernous sinus thrombosis

  Causes of bilateral ptosis: Myasthenia gravis, snake bite, botulism, bilateral Horner syndrome, and incomplete reversal of muscle relaxant
- *Size of the pupil:* Look for miosis and mydriasis. Asymmetrical pupil always indicates neurological problem.

  Causes of miosis include opioid overdose, organophosphates poisoning, cholinesterase inhibitors, pontine hemorrhage, clonidine toxicity, and cholinergic toxicity

  Causes of mydriasis include anticholinergic agents, tricyclic antidepressants, amphetamines,

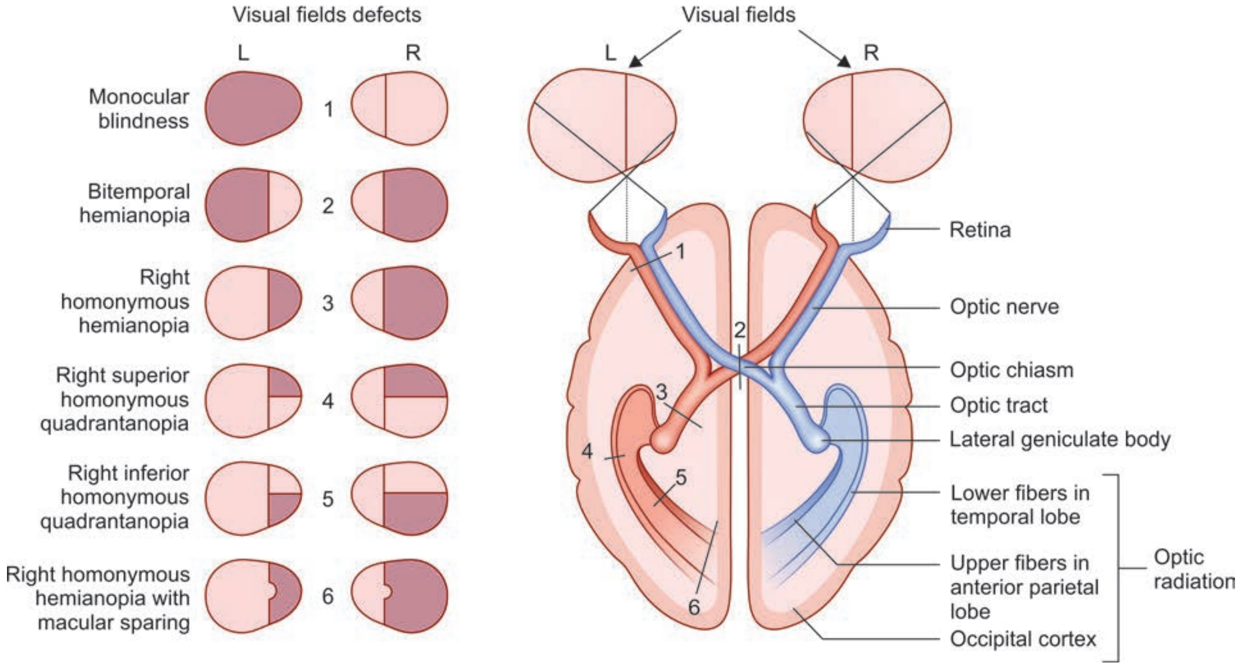
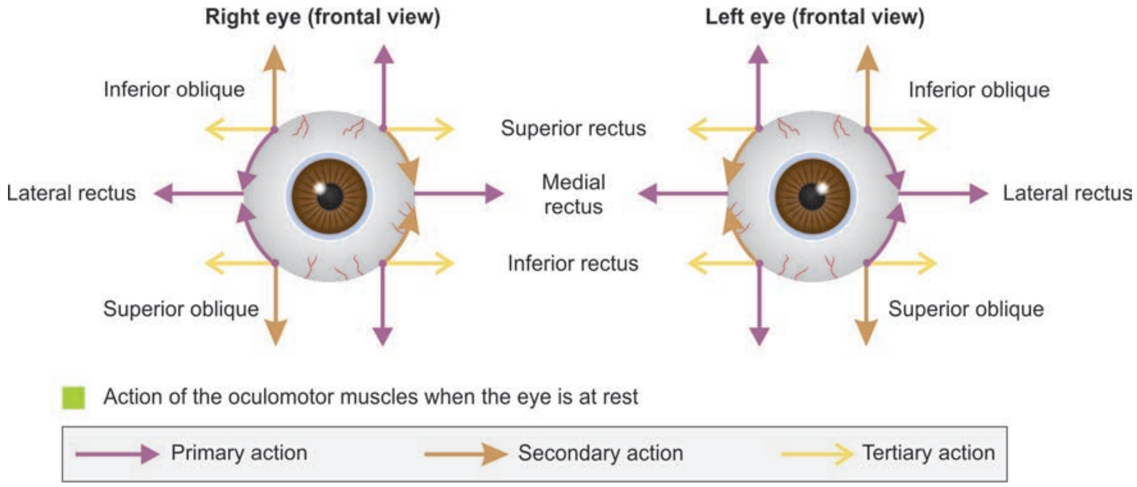

Visual fields defects
L
R
Monocular blindness
1
Bitemporal hemianopia
2
Right homonymous hemianopia
3
Right superior homonymous quadrantanopia
4
Right inferior homonymous quadrantanopia
5
Right homonymous hemianopia with macular sparing
6
Visual fields
L
R
Retina
Optic nerve
Optic chiasm
Optic tract
Lateral geniculate body
Lower fibers in temporal lobe
Upper fibers in anterior parietal lobe
Occipital cortex
Optic radiation
1
2
3
4
5
6

Fig. 1: Various field defects depending on the site of the lesion.



Upward to the right—right SR and left IO	Mid upward—both SR recti and both IO	Upward to the left—left SR and right IO
To the right—right LR and left MR	Primary position—contraction of all extraocular muscles	To the left—left LR and right MR
Right down—right IR and left SO	Mid down—both IR and both SR	Left down—left IR and right SO

Fig. 2: Action of oculomotor muscles. (IO: inferior oblique; IR: inferior rectus; LR: lateral rectus; MR: medial rectus; SO: superior oblique; SR: superior rectus)

3,4-methylenedioxymethamphetamine (MDMA), lysergic acid diethylamide (LSD), cocaine, central herniation, midbrain injury, and brain death.

- *Diplopia:* It means double vision. If diplopia occurs only if both eyes open, it is called binocular diplopia.

  Uniocular diplopia usually indicates intraocular issues such as ectopia lentis, astigmatism, lens opacities, corneal opacities, and vitreous opacities.

  Binocular diplopia indicates extraocular muscle weakness. Defective movement of the affected eye results in the image of the object falling on two different points on the retinae of the two eyes. A simple way to detect the weak eye is to see which eye closure causes the lateral or false image to disappear. This is the weak eye.
- *Conjugate gaze palsy:* Upper motor neuron lesion of the III, IV, and VI CN leads to conjugate gaze palsy. Lateral conjugate palsy is seen in the pontine and frontal lobe lesion, but in frontal lobe lesion eyes deviate to the side of the lesion, whereas in pontine lesion eyes deviate to the opposite side of the lesion. Upward conjugate gaze palsy is seen in the lesion of the midbrain at the level of superior colliculus, and downward conjugate gaze palsy is seen in the lesion of the midbrain at the level of inferior colliculus.
- *Internuclear ophthalmoplegia:* Internuclear ophthalmoplegia occurs due to lesion of medial longitudinal fasciculus (MLF), which connects ipsilateral third nerve nucleus in the midbrain with contralateral sixth nerve nucleus in the pons. Internuclear ophthalmoplegia is characterized by absence of adduction of eye on the side of MLF lesion with mild weakness of abduction with nystagmus on the contralateral side. Lesion in the midbrain is associated with loss of conversion, which is preserved in pontine lesion. Common causes of the lesion are infarction and multiple sclerosis.
- *Nystagmus:* Nystagmus is conjugate involuntary rhythmic oscillation of the eyes. Two types of nystagmus are pendular and jerky nystagmus.

  Pendular nystagmus is characterized by rapid horizontal oscillation of equal amplitude on either side of midline on forward gaze. Visual defects from infancy result in pendular nystagmus.

  Jerky nystagmus is characterized by horizontal oscillation of unequal amplitude on either side of midline, with slow drift in one direction and fast corrective phase in opposite direction, fast phase determines the direction of the nystagmus.

Horizontal, vertical, optokinetic, see saw, upbeat, and downbeat nystagmus are the types of jerky nystagmus.

Fifth CN (trigeminal nerve): Sensation over the face received by trigeminal nuclei from the ophthalmic, maxillary, and mandibular division of the trigeminal nerve.

Corneal and conjunctival reflex is tested with wisp of the cotton. Afferent is trigeminal nerve and efferent is facial nerve.

Nuclear lesion of fifth nerve: Diseases affecting pons, medulla, and upper cervical cord up to C2. Common diseases are stroke, demyelination, tumor, syringomyelia, or syringobulbia.

Lateral medullary syndrome due to occlusion of the posterior inferior cerebellar artery is characterized by ipsilateral loss of pain and temperature over the face, ipsilateral Horner syndrome, ipsilateral cerebellar signs and symptoms of vertigo, nausea, and vomiting, and contralateral hemianesthesia.

Postganglionic nerve lesions: Commonly seen in cavernous sinus lesion (associated with ipsilateral III, IV, and VI CN palsy), Gradenigo syndrome (associated with ipsilateral VI nerve palsy and retroorbital pain), and superior orbital fissure syndrome (associated with III, IV, and VI nerve palsy).

Seventh CN (facial nerve): UMN palsy of the facial nerve causes paralysis of contralateral lower half facial muscle, lower motor neuron (LMN) facial palsy causes ipsilateral paralysis of all facial muscles because upper half of face has bilateral presentation compared to the lower half, which has unilateral presentation.

Facial nerve has sensory and motor functions. Its lesion causes deviation of mouth to normal side, inability to raise eyebrow and close eyes, leaking of air on paralyzed side while blowing the air, and inability to purse and whistle. Facial nerve lesion causes loss of taste sensation over anterior two-thirds of the tongue on the ipsilateral side.

Table 2 shows the various causes of a facial nerve palsy. **Table 3** shows the difference between an upper and lower motor VII nerve palsy. **Table 4** shows the localization of the VII nerve palsy.

The eighth CN (vestibulocochlear nerve): Eighth CN has two components—vestibular and cochlear. Cochlear nerve is responsible for hearing and vestibular nerve is concerned with balance and equilibrium.

TABLE 2: The various causes of a facial nerve palsy.

Unilateral	***Bilateral***
• *UMN type:* Stroke, tumor, demyelination • *LMN type:* Bell's palsy, Ramsay Hunt syndrome, parotid tumor, head injuries, skull base tumors, diabetes	• *UMN type:* Multi-infarct dementia, motor neuron disease • *LMN type:* Guillain-Barré syndrome, leukemia/lymphoma, sarcoidosis-uveoparotid fever

(LMN: lower motor neuron; UMN: upper motor neuron)

TABLE 3: Difference between an upper and lower motor VII nerve palsy.

Bilateral UMN palsy	***Bilateral LMN palsy***
Bell's phenomenon—absent	Bell's phenomenon—present
Emotional fibers—spared	Emotional fibers—affected
Corneal reflex—present	Corneal reflex—absent
Associated with long tract signs	Long tract signs absent
Jaw jerk—exaggerated	Jaw jerk—normal

(LMN: lower motor neuron; UMN: upper motor neuron)

Hearing can be tested with a tuning fork or by using speech in one ear with the sound blocked or masked in the other ear.

Vestibular nerve function is tested with Caloric test, oculo-cephalic reflex or dolls eye movement. Derangement of vestibular function usually causes vertigo, dizziness, nausea, and unsteady gait.

Oculocephalic reflex—examiner should briskly move the head to one side and look for eye movement. Normally both eyes move to the opposite side. In brain death, there should be no movement of the eyes relative to the head movement.

Caloric testing normal ear response to a cold temperature is nystagmus to opposite direction and warm temperature nystagmus is toward the same ear. In brain death, this response is absent.

The ninth and tenth CN (glossopharyngeal and vagus nerves): Nerve function is tested with taste sensation over posterior one-third of the tongue, palatal reflex, and gag reflex.

In unilateral palatal palsy, soft palate on the side of the lesion remains lower than normal side and uvula gets

TABLE 4: Localization of the lesions causing VII nerve palsy.

Site of lesion	***Type of lesion***	***Associated clinical feature***
Cortex	• Cerebral infarction • Hemorrhage • Tumor	Contralateral facial weakness mainly of lower face often associated with hemiparesis on the same side
Pons	• Infarction • Demyelination • Hemorrhage • Tumor	LMN type of ipsilateral face weakness may be associated with VI involvement; contralateral hemiparesis
Cerebellopontine angle	• Acoustic neuroma • Meningioma	LMN type of ipsilateral face weakness; deafness and tinnitus; ophthalmic division of V nerve may be affected
Facial canal	• Bell's palsy • Mastoiditis • Herpes-zoster (Ramsay Hunt syndrome)	LMN type of ipsilateral face weakness ± loss of taste salivation and lacrimation, if lesion is proximal to chorda tympani ± hyperacusis if lesion is proximal to nerve to stapedius
Parotid gland	• Tumor • Sarcoidosis	Selective weakness of parts of face due to branch involvement
Neuromuscular junction	Myasthenia gravis	Associated ptosis and external muscle weakness, dysphagia, dysarthria, ± limb weakness
Muscles	• Muscular dystrophy • Myositis	Associated with limb muscles weakness and tenderness

(LMN: lower motor neuron)

deviated to the normal side. In bilateral palatal palsy, the palate remains immobile on both sides.

TABLE 5: Causes of multiple cranial nerve (CN) palsies.

Site of lesion	***Cranial nerves involved***	***Common causes***
Cavernous sinus	III, IV, VI CN and first branch of V with proptosis	Aneurysm or thrombosis of cavernous sinus, invasive lesion from paranasal sinuses or Sella turcica
Sphenoid fissure	III, IV, VI, first division of V	Invasive tumor of sphenoid bone
Retrosphenoid space	II, III, IV, V, VI	Large tumor of middle cranial fossa
Apex of petrous bone	V, VI	Petrositis, tumor of the petrous bone
Internal auditory meatus	VII, VIII	Acoustic neuroma and tumor of petrous bone
Cerebellopontine angle	V, VII, VIII, sometime IX	Acoustic neuroma, meningioma
Jugular foramen	IX, X, XI	Tumor and aneurysm
Posterior lateral condylar space	IX, X, XI, XII	Tumor of parotid gland, carotid body, metastatic tumor
Posterior retroparotid space	IX, X, XI, XII and Horner's syndrome	Tumor of parotid gland, carotid body, metastatic tumor, tuberculous adenitis

Gag reflex is tested by tickling the posterior wall of the pharynx and noting the reflex contraction of the pharynx thus produced. Gag reflex is absent on the side of the lesion of the ninth and tenth CN (lower motor neuron type of palsy). Exaggerated gag reflex is seen in pseudobulbar palsy (upper motor neuron type of palsy). Absent gag reflex is important in brain death evaluation.

Eleventh CN (accessory nerve): This is a pure motor nerve supplying the sternocleidomastoids and the trapezius muscles.

The twelfth CN (hypoglossal nerve): To examine it, the patient is asked to put out his tongue and look for the wasting, deviation, fibrillation, tremors, or any other abnormal movement such as chorea of the tongue.

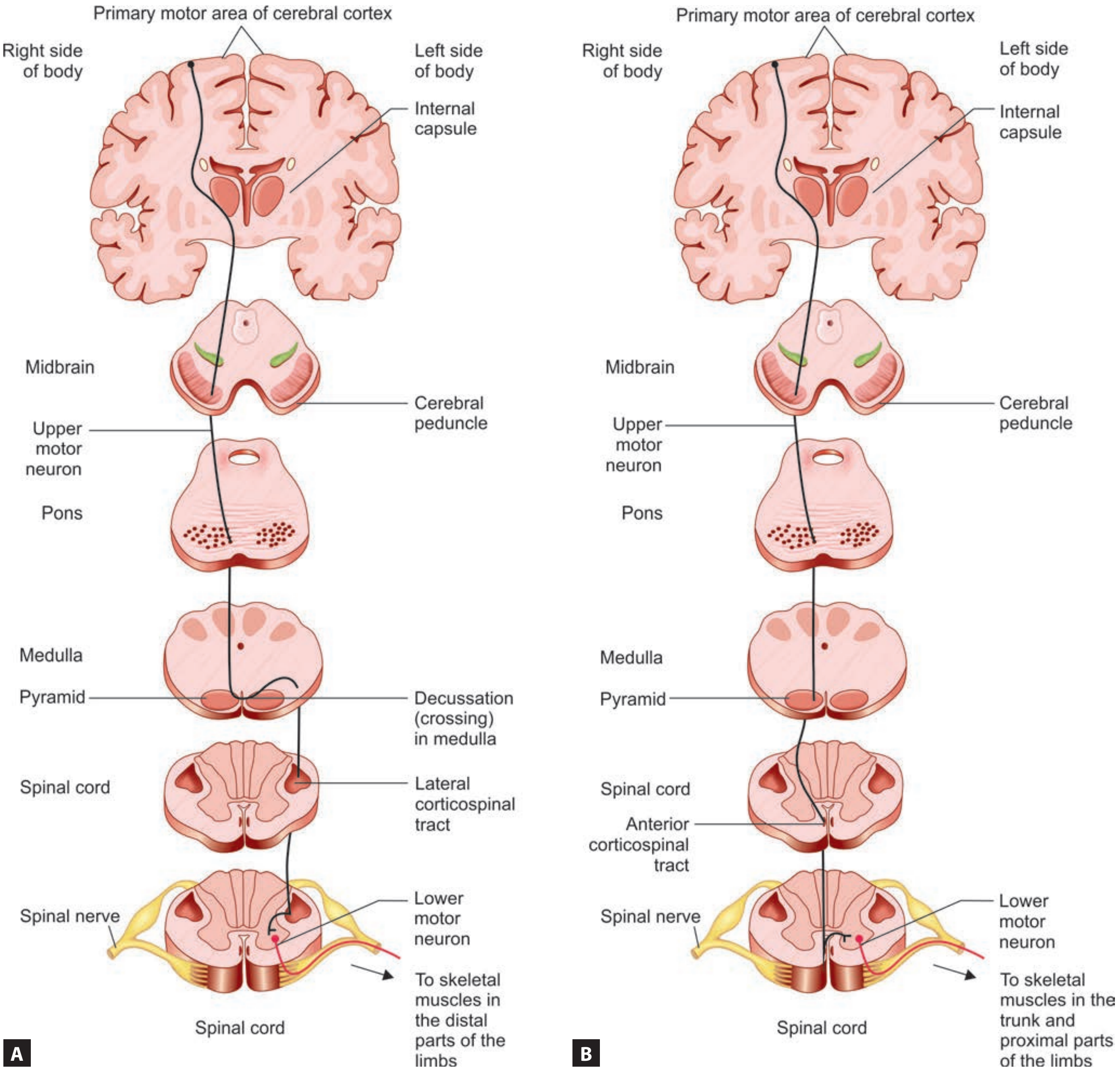
Table 5 shows causes of multiple cranial nerve palsies.

The Motor System

The motor system is primarily involved in smooth and coordinated voluntary movements. The components of the motor system are the corticobulbar and corticospinal (UMN), basal ganglia and cerebellum, and the neuromuscular system (LMN). Diseases of the motor system cause weakness (paresis and paralysis), involuntary movement, and ataxia.

The anatomical details of the corticospinal tracks are shown in **Figures 3 and 4**. The anatomical features of the extrapyramidal system are shown in **Figure 5**.

Patients presenting with neurological weakness need to be evaluated for a diagnosis. In frail or long-term patients, there is a generalized weakness, but this is usually generalized and mild in terms of its grade. When evaluating weakness in an ICU patient, one specifically looks for ocular or facial weakness, neck weakness, and limb weakness. One also evaluates for proximal versus distal weakness.


Primary motor area of cerebral cortex
Right side of body
Left side of body
Internal capsule
Midbrain
Cerebral peduncle
Upper motor neuron
Pons
Medulla
Pyramid
Decussation (crossing) in medulla
Spinal cord
Lateral corticospinal tract
Spinal nerve
Lower motor neuron
To skeletal muscles in the distal parts of the limbs
Spinal cord
A
Primary motor area of cerebral cortex
Right side of body
Left side of body
Internal capsule
Midbrain
Cerebral peduncle
Upper motor neuron
Pons
Medulla
Pyramid
Spinal cord
Anterior corticospinal tract
Spinal nerve
Lower motor neuron
To skeletal muscles in the trunk and proximal parts of the limbs
Spinal cord
B

Figs. 3A and B: Anatomy of the corticospinal tracks. (A) Lateral corticospinal pathway; (B) Anterior corticospinal pathway.

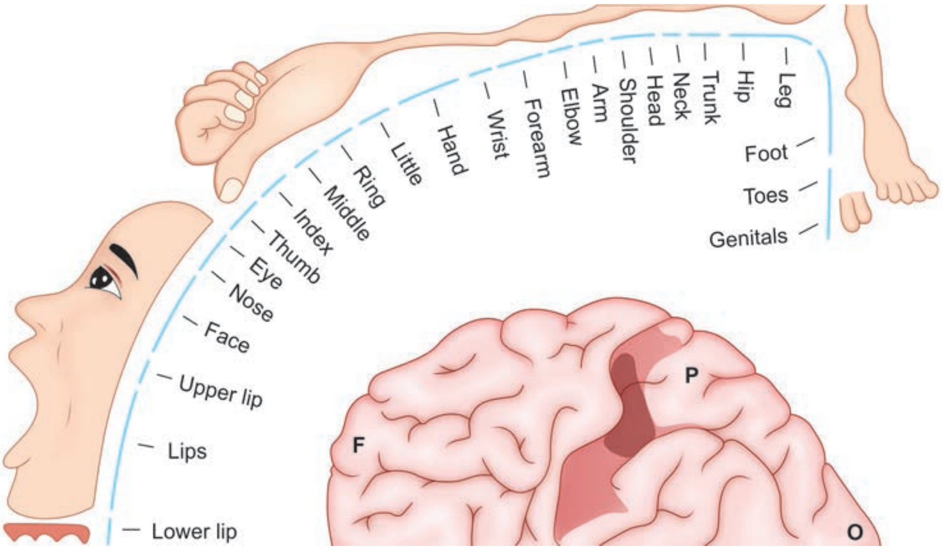
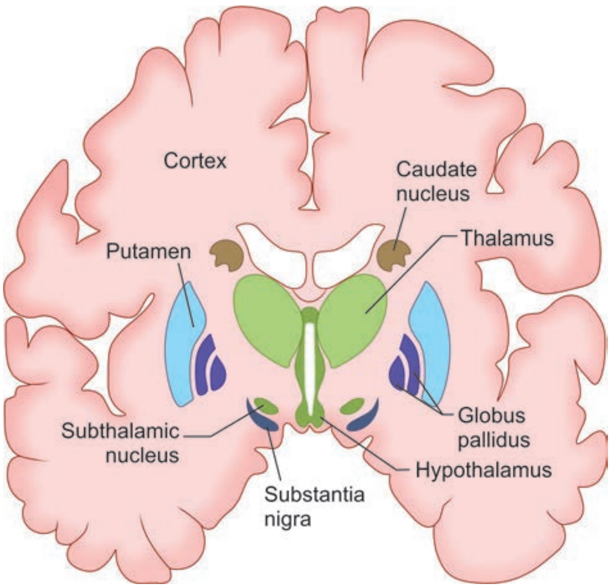

Leg
Hip
Trunk
Neck
Head
Shoulder
Arm
Elbow
Forearm
Wrist
Hand
Little
Ring
Middle
Index
Thumb
Eye
Nose
Face
Upper lip
Lips
Lower lip
Foot
Toes
Genitals
P
F
O

Fig. 4: Somatic representation of the corticospinal tracks (P: parietal lobe; F: frontal lobe; O: occipital lobe).


Cortex
Caudate nucleus
Thalamus
Putamen
Subthalamic nucleus
Globus pallidus
Hypothalamus
Substantia nigra

Site of lesion	***Sign***
Substantia nigra, red nucleus	Resting tremor
Substantia nigra, putamen	Muscular rigidity
Substantia nigra, globus pallidus, putamen	Hypokinesia
Caudate nucleus	Chorea
Subthalamic nucleus	Hemiballismus
Putamen	Dystonia, athetosis

Fig. 5: The anatomical features of the extrapyramidal system.

Asymmetrical weakness is usually due to a CNS illness, usually a stroke. Occasionally, it may be due to a nerve plexus injury. Symmetrical weakness is usually due to a nerve involvement such as Guillain-Barré syndrome (GBS) or a myoneural one such as myasthenia gravis. Other causes for this weakness include toxins, drugs (including residual effect of paralyzing drugs in renal failure), and occasionally following a snake bite.

From an ICU perspective, one must be vigilant for respiratory muscle weakness and bulbar weakness, as these can put patients at risk of aspiration and hypoventilation. If these are undetected, or detected late, they can quickly become life-threatening. In patients with respiratory muscle weakness, monitoring the forced vital capacity (FVC), peak expiratory flow rate (PEFR), or single breath count allows early detection of impending ventilatory failure.

The corticobulbar and corticospinal (pyramidal) system: The UMN arises partly from nerve cells in the precentral motor cortex of the cerebrum and some of these neurons arise from the giant Betz cells. Nerve fibers arising from these cortical cells (including Betz cells) then come together in the corona radiata and converge in the internal capsule. From internal capsule, it travels through the cerebral peduncle to enter the midbrain. In the brainstem, the tract gives off corticobulbar fibers, which travel to the contralateral motor nuclei of the CN. Most fibers of the pyramidal tract decussate at lower part of the medulla to form the crossed pyramidal tract, which descends in the lateral column of the spinal cord on the opposite side. A small proportion of uncross fibers continue downward in the anterior column, forming the uncrossed pyramidal tract, which travels downward only up to the dorsal spinal cord and they supply the axial muscles. Fibers of the pyramidal tract synapse with the anterior horn cells through internuncial neurons in the gray matter of the spinal cord. The LMN arises from the anterior horn cells.

Examination of the Spinomotor System

The spinomotor system should be examined systematically in the following order:

1. Nutrition
2. Tone
3. Power
4. Coordination
5. Involuntary movements
6. Gait

Table 6 shows the differentiation between spasticity and rigidity. **Table 7** shows the grading of power loss. **Table 8** shows the innervation of the muscle groups. **Table 9** shows the difference between an upper motor and lower motor lesion.

Table 10 shows the clinical features and differential diagnosis of lower motor and other peripheral nervous system weakness.

Nutrition: Change in nutrition of muscle groups detected by comparing with its fellow on the normal side. Muscle

TABLE 6: The differentiation between spasticity and rigidity.

Spasticity	*Rigidity*
It is a sign of pyramidal tract lesion	It is sign of extrapyramidal tract lesion
Tone is more in antigravity muscle compared to gravity assisted muscle	Tone is equal in both antigravity and gravity assisted group of muscle
Clasp knife type of spasticity	Lead pipe or cogwheel type of rigidity
Velocity dependent	Not a velocity dependent
Abdominal reflex is lost	Abdominal reflex preserve
Deep tendon reflex is exaggerated	Deep tendon reflexes are preserved
Plantar is extensor	Normal plantar
Clonus present	Clonus absent

TABLE 7: The grading of power loss.

Grade 0	Total paralysis
Grade I	There is a visible or palpable flicker of contraction, but no resultant movement of joint
Grade II	Movement on eliminating gravity (lateral movements in bed)
Grade III	Movement against gravity
Grade IV	Movement against gravity and some resistance
Grade V	Normal power

TABLE 8: The innervation of the major muscle groups.

Muscle group	*Peripheral nerve*	*Main segmental nerve supply*
Shoulder		
Supraspinatus	Suprascapular	C5
Infraspinatus	Suprascapular	C5
Deltoid	Circumflex	C5
Serratus anterior	Nerve to serratus anterior	C5, C6, C7
Pectoralis major	Pectoral nerves	C5, C6, C7, C8
Elbow		
Biceps	Musculocutaneous	C5
Brachioradialis	Radial	C5, C6
Triceps	Radial	C7
Wrist		
Flexor carpi radialis and flexor carpi ulnaris	Median and ulnar	C6, C7, C8
Extensor carpi radialis longus and extensor carpi ulnaris	Radial	C6, C7
Fingers		
Flexor digitorum profundus	Median and ulnar	C8
Extensor digitorum communis	Posterior interosseous	C7, C8
Abductor pollicis brevis	Median	C8, T1
Extensor pollicis brevis	Radial	C8
Extensor pollicis longus	Radial	C8
Flexor pollicis longus	Median	C8
Opponens pollicis	Median	T1
Adductor pollicis brevis	Median	T1
Adductor pollicis	Ulnar	T1

Contd…

Contd…

Muscle group	***Peripheral nerve***	***Main segmental nerve supply***
Lumbricals and interossei	*Median nerve:* Lumbrical 1 and 2nd *Ulnar:* 3rd and 4th lumbrical, all interossei	C8, T1
Hip		
Ilio-psoas	Femoral	L1, L2, L3
Gluteus maximus	Inferior gluteal	L5, S1
Gluteus medius and minimus	Superior gluteal	L4–S1
Adductor longus	Obturator	L2, L3
Knee		
Quadriceps femoris	Femoral	L3, L4
Hamstrings	Sciatic	L4, L5, S1, S2
Ankle		
Tibialis anterior	Anterior tibial	L4, L5
Tibialis posterior	Medial popliteal	L4
Peronei	Musculocutaneous	L5, S1
Gastrocnemius	Medial popliteal	S1
Toes		
Extensor digitorum longus	Anterior tibial	L5
Flexor digitorum longus	Medial popliteal	S1, S2
Extensor hallucis longus	Anterior tibial	L5

wasting can generalize in systemic diseases or localized to particular muscle group due to LMN lesion or muscle diseases.

Tone: Tone is resistance offered to passive movement.

Hypertonia: Resistance felt on passive movements of the joints, either in the form of spasticity or rigidity.

Clasp knife spasticity resistance is felt only at the beginning or at the end of the passive movement.

In lead pipe rigidity, there is uniform resistance offered to passive movement, commonly seen in parkinsonism, basal ganglia neoplasms, and catatonia.

In cogwheel rigidity, resistance to passive movement is interrupted by alternate contractions of the agonist and antagonist muscles due to presence of associated tremor, commonly seen in Parkinson disease and carbon monoxide poisoning.

TABLE 9: The clinical features and differential diagnosis of lower motor and other peripheral nervous system weakness.

Signs of pyramidal tract (upper motor neuron) lesion	***Signs of motor unit or lower motor neuron lesion***
• No muscle wasting but muscle wasting can occur in the late stages due to disuse atrophy • Increased muscle tone • Paralysis of voluntary movement; early, distal weakness involving fine movements of the hand and weakness is more in extensors of the upper limbs and flexors of the lower limbs; group of muscles first affected are the last to recover • Absent abdominal reflex • Positive Babinski sign, i.e., extensor plantar response • Brisk or exaggerated deep tendon reflexes • Sustained clonus	• Wasting of the affected muscles • Hypotonia • Weakness or paralysis of muscles supplied by the affected anterior horn cells • Fasciculation (sign of degenerating anterior horn cells or irritative lesions of the nerve roots or peripheral nerves) • Flexor/absent plantar response • Reduced or absent deep tendon reflexes

Hypotonia: It is characterized by decreased resistance to passive movement of the joints, results in widening the range of movement at the joint and inability to maintain posture. On inspection, hypotonic muscles are lax and assume a pendulous shape when allowed to hang freely. On palpation, muscle feels flabby.

Lower motor lesions cause marked hypotonia. It can also be seen in neuronal shock in UMN disease, cerebellar disease, and rheumatic chorea.

Power: Power is resistance offered to active movement.

Power is force of contraction generated by voluntary movement. It should be tested in different muscle groups from head to foot. Any weakness suspected should be compared with opposite normal side.

Coordination: Detailed coordination testing is not usually feasible in critically ill patients. Coordination of limb can be tested effectively only if power is greater than grade III.

Tests done to evaluate coordination of the lower limb are Romberg's test, tandem walking, knee-heel test, and tests done to evaluate coordination of the upper limb are the finger-nose test, finger-finger-nose test, and dysdiadochokinesis.

TABLE 10: The clinical features and differential diagnosis of lower motor and other peripheral nervous system weakness.

	Anterior horn cell	*Root/radicles*	*Peripheral nerve*	*Myoneural junction*	*Muscle disease*
Distribution of the weakness	Can be proximal or distal group of muscle weakness, sometime neck flexors get involved	Asymmetrical weakness, which is distal along the nerve distribution	Symmetrical weakness, which is distal along the nerve distribution	Proximal muscle weakness; there may be involvement of bulbar, neck, eye, and respiratory muscle	Proximal muscle weakness
Wasting	Marked	Present	Present-moderate	Absent	Present
Fasciculations	Present and marked	Absent	May be present	Absent	Absent
Sensory loss	Absent	Present	Present	Absent	Absent
Fatigue	Mild	Mild	Mild	Severe	Mild to moderate
Reflexes	Absent	Absent	Absent	Preserve	Preserve

Involuntary movements: Involuntary movement occurs due to involvement of extrapyramidal system. Common involuntary movements are tremors, myoclonus, fasciculation, dystonia, hemiballismus, chorea, and athetosis.

Tremors: These are rhythmical and oscillatory movements caused by regular, rhythmical, contractions of the agonist and antagonist muscles.

Flapping tremors are seen in hepatic failure, renal failure, and respiratory failure.

Rest tremor seen in parkinsonism.

Action/postural tremors are seen when limb is active and present throughout the range of movement; common causes are familial, anxiety, hyperthyroidism, delirium tremens, cerebellar lesion, Wilson disease, sympathomimetic drugs (salbutamol), antidepressant, lithium, and sodium valproate.

Intentional tremors occur when limb reaches its target. Common causes include lesion of the cerebellum and its connection.

Myoclonus: It is repetitive and sometimes rhythmical, rapid, brief shock-like jerky movement of the muscle. It may involve whole muscle or a small number of muscle fibers contraction. Sometime contraction may be too slight to cause movements or may cause violent movements.

Causes of the generalized myoclonus are uremia, hyponatremia, hypocalcemia, hepatic failure, CO_2 narcosis, hypoxic ischemic brain damage, drug-induced myoclonus, alcohol and drug withdrawal, and post-traumatic myoclonus, and other rare causes include subacute sclerosing leukoencephalitis, Alzheimer disease, and spinocerebellar degeneration.

Fasciculation: It is irregular, nonrhythmical contraction of muscle fascicles. Conditions causing fasciculation are motor neuron disease, organophosphorus poisoning, thyrotoxic myopathy, carcinomatous myopathy, and poliomyelitis.

Gait: Most of the time, it may not be feasible to assess the gait in critically ill patients because patients may be too sick. Common abnormal gait is hemiplegic gait, spastic gait, high stepping gait, stamping gait, ataxic gait, shuffling gait, and waddling gait.

Sensory System

It is rare that a detailed sensory examination is needed in an ICU or emergency patient. Usually, evaluating the response to pain, touch, and occasionally position or vibration testing are all that are required.

The findings of the sensory examination are usually complementary to those of the motor examination, and together, they are used to make or confirm the diagnosis of “where is the lesion and what is the lesion.”

Sensation is divided into the following:

- *Superficial:* It includes pain and temperature, which is carried by spinothalamic tract.
- *Deep:* It includes joint position, vibration sense carried by dorsal column pathway.
- *Cortical:* It can be tested only when other sensations are intact.

A more detailed examination of the sensory system includes testing of touch, pain, temperature, joint position, joint sense, and vibration sense. Cortical sensation routinely not tested in critically ill patients: tactile localization, tactile extinction, or sensory inattention, two-point discrimination, stereognosis, and graphesthesia.

Dissociative sensory loss: Pain and temperature sensation (spinothalamic tract lesion) are lost but touch sensation (posterior column) is preserved. Common causes are intramedullary lesions (hydromyelia or syringomyelia), anterior spinal artery thrombosis, Pica syndrome, Brown–Sequard syndrome, leprous neuritis, amyloid neuropathy, and diabetic polyneuropathy.

The details of the sensory system are presented in **Figures 6 to 8**.

Sensory pathways: From peripheral nerve, sensation travels to the dorsal roots and then to the dorsal horn of spinal cord, from there depending on the type of sensation travel by either spinothalamic or posterior column (lemniscal system).

Posterior column pathway (lemniscal system): Fine touch, joint position, joint sense, and vibration are carried by the ipsilateral posterior column pathway, which ascends till the gracile and cuneate nuclei of the lower medulla. Second-order neuron decussates and ascends in the medial lemniscus located medially in the medulla and in the tegmentum of pons and midbrain and synapses in ventroposterolateral nucleus (VPL) of the thalamus. Third-order neuron carries sensation to the parietal cortex.

Spinothalamic system: Small fiber carries pain, temperature, and crude touch sensation, which ascend for 2–3 segments and cross and ascend in the lateral spinothalamic tract through spinal cord, brainstem to VPL of the thalamus, and from there to postcentral gyrus of parietal cortex (area 3, 1, 2).

Reflexes

Reflexes are involuntary adaptive response to the stimulation of a sense organ.

A detailed examination of all the reflexes is rarely needed in an ICU patient, especially one with preserved consciousness and normal higher functions. The reflexes are either the CN reflexes, the superficial reflexes, and the deep tendon reflexes (DTR). One should be familiar with the innervation of these reflexes.

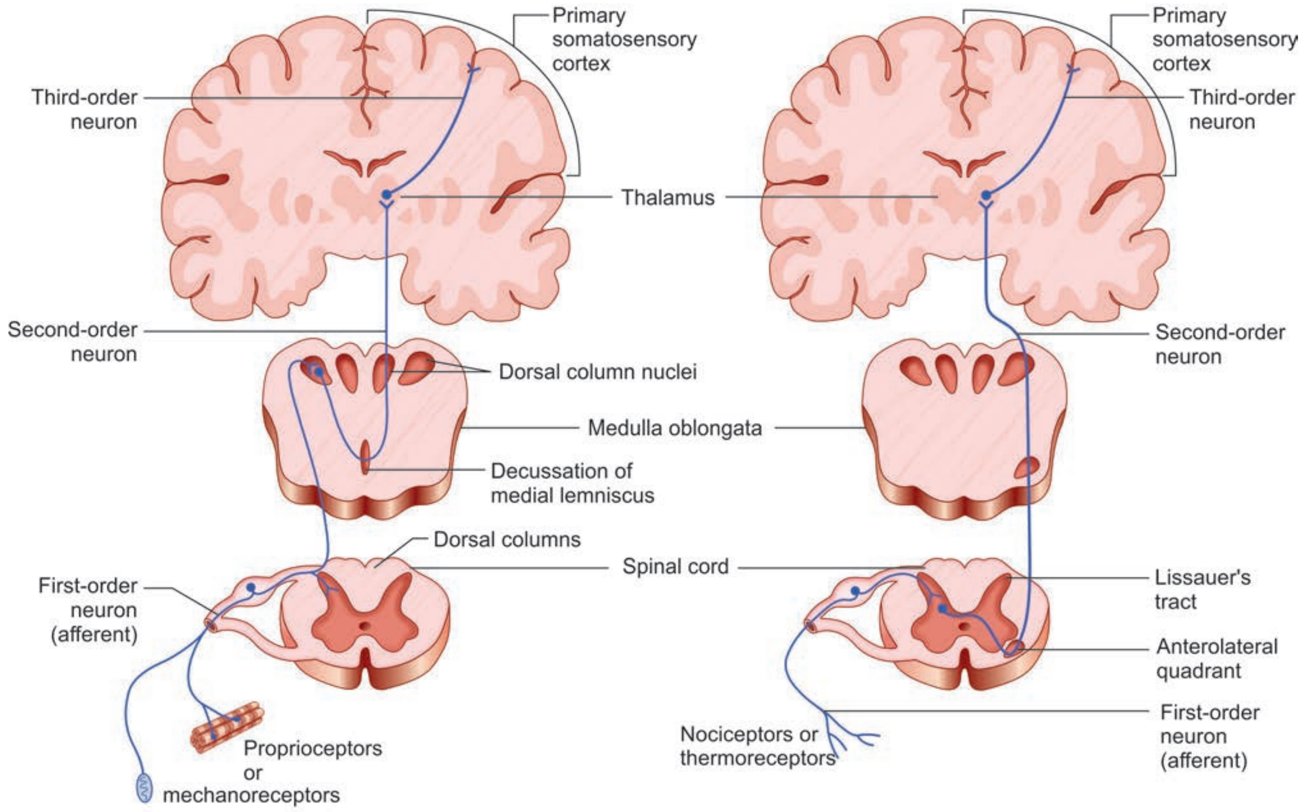

Primary somatosensory cortex
Third-order neuron
Thalamus
Second-order neuron
Dorsal column nuclei
Medulla oblongata
Decussation of medial lemniscus
Dorsal columns
Spinal cord
First-order neuron (afferent)
Proprioceptors or mechanoreceptors
Primary somatosensory cortex
Third-order neuron
Second-order neuron
Lissauer's tract
Anterolateral quadrant
First-order neuron (afferent)
Nociceptors or thermoreceptors

Fig. 6: Anatomical details of the sensory fibers.

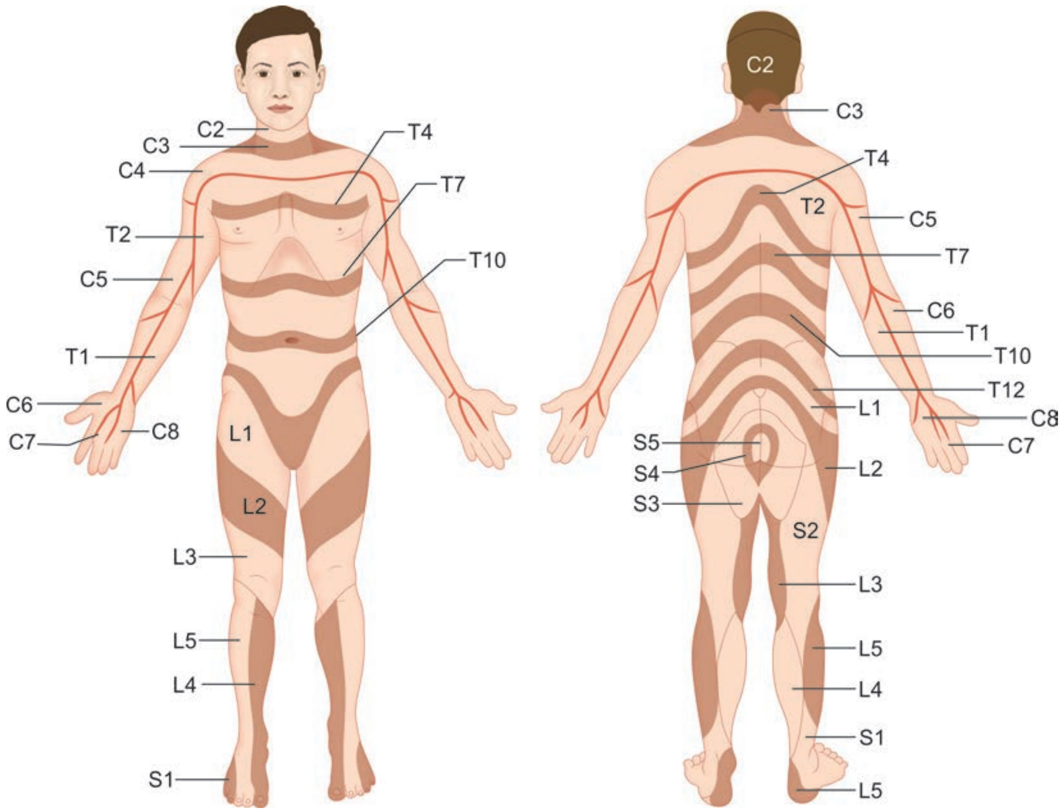


Fig. 7: Sensory dermatomes.

The plantar reflex is very useful for detecting a neurological disease. An upgoing plantar signifies an upper motor involvement and a unilateral upgoing plantar suggests intracranial disease, though it can also be observed in hemispinal lesions.

Diminished or absent tendon reflexes: Seen in LMN lesions involving any part of the reflex arc.

Exaggerated tendon reflexes: Pyramidal tract lesion results in exaggerated DTR. Reflexes may be brisk if the patient is agitated, frightened, or anxious.

Table 11 shows the cranial and superficial reflexes. **Table 12** shows the grading of the DTR. **Table 13** shows the segmental innervation of the reflexes.

To test patient for DTR, proper patient position is very important. Patient should lie down comfortably in a partially propped up position. Long and flexible knee hammer should be used to strike the muscle tendon.

CASE SCENARIOS: OUTCOME

Case Scenario 1

Patient presented with new onset aphasia and hemiparesis.

An MRI brain with a stroke protocol was done. The patient was in the window period of 3 hours. The patient was offered the options of thrombolysis or intervention and he opted for the former. The patient was observed in the ICU for 24 hours and then transferred to the floors. He received standard cerebrovascular accident (CVA) therapy including antiplatelet agents, statins, and deep vein thrombosis (DVT) prophylaxis, followed by physiotherapy, speech therapy, and rehabilitation.

(*Note:* The use of thrombolytics or intervention is primarily to improve the functional outcomes at 3 months and beyond. Most studies have failed to show a benefit in terms of rapid reversal of neurological deficit or mortality.)

Case Scenario 2

Patient presented with progressive weakness, bilateral facial palsy, and absent reflexes following COVID-19 infection.

An electrophysiologic study (EPS) of nerve conduction velocity (NCV) was done that confirmed the diagnosis of GBS or acute inflammatory demyelinating polyneuropathy (AIDP) secondary to COVID-19. He was offered the

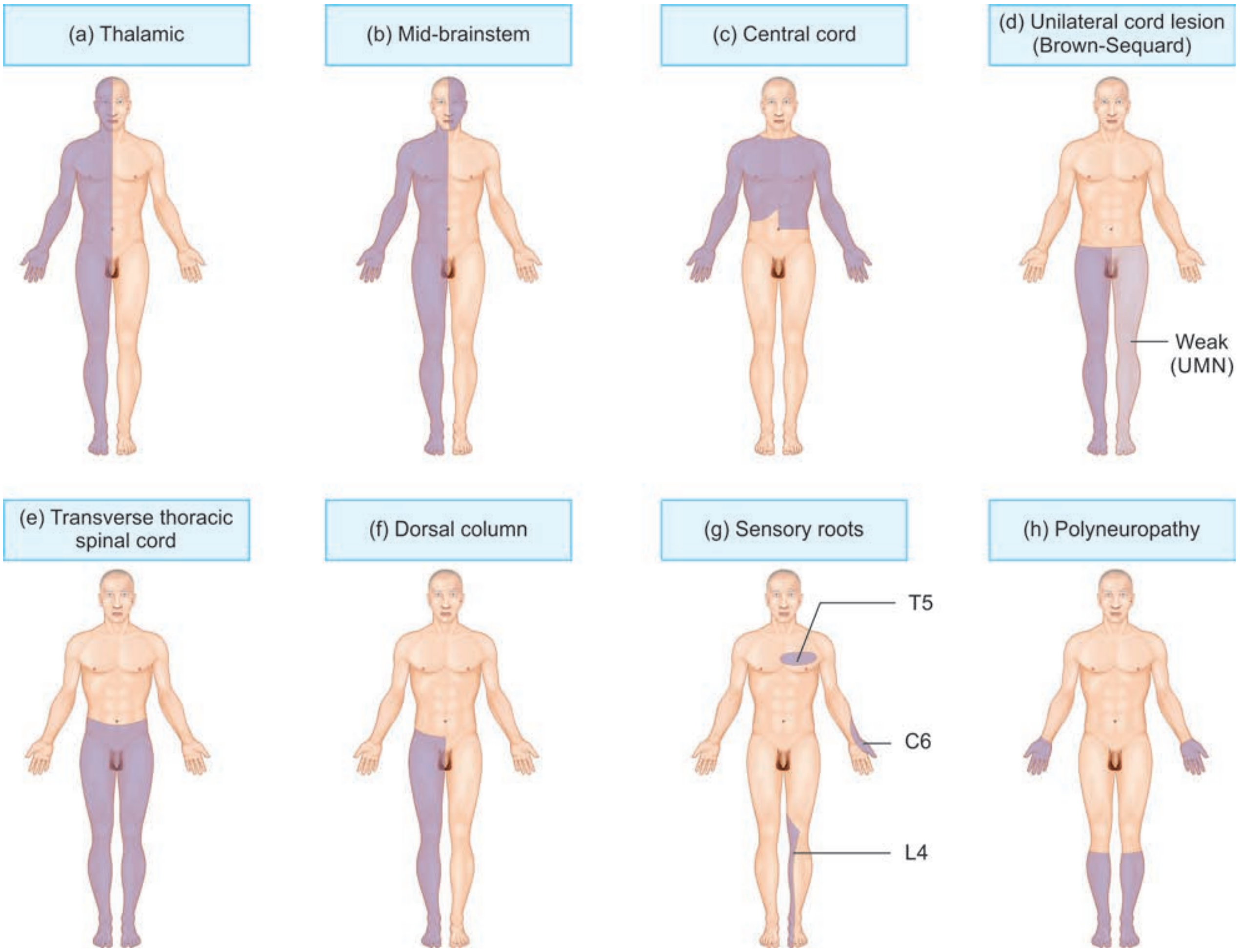

(a) Thalamic
(b) Mid-brainstem
(c) Central cord
(d) Unilateral cord lesion (Brown-Sequard)
Weak (UMN)
(e) Transverse thoracic spinal cord
(f) Dorsal column
(g) Sensory roots
T5
C6
L4
(h) Polyneuropathy

Fig. 8: Distribution of sensory loss as per site of anatomical lesion. (UMN: upper motor neuron)

TABLE 11: The cranial and superficial reflexes.

Reflex	***Segmental innervation***	***Normal response***
Corneal and conjunctival	• *Afferent:* V nerve • *Center:* Pons • *Efferent:* VII nerve	Brisk closure of the eyes
Pharyngeal/ Gag reflex	• *Afferent:* IX nerve • *Center:* Medulla • *Efferent:* X nerve	Contraction of the pharyngeal muscles
Cough reflex	• *Afferent:* Vagus (X) nerve • *Center:* Medulla • *Efferent:* Phrenic nerve	Coughing
Plantar reflex	• *Afferent:* Tibial nerve • *Segment:* L5, S1, S2 • *Efferent:* Tibial nerve	Normally, the great toe will flex at the metatarsophalangeal joint, accompanied by flexion of the other toes

TABLE 12: The grading of the deep tendon reflexes.

Grade 0	Absent reflexes
Garde 1	Present (as normal ankle jerk)
Grade 2	Brisk (as normal knee jerk)
Grade 3	Exaggerated
Grade 4	Clonus

options of intravenous immunoglobulin (IVIG) or plasma exchange and opted for the latter as it was significantly cheaper. He was observed initially in the ICU and then managed on the floors. He made a slow recovery and was discharged after a week.

Case Scenario 3

Patient presented with COPD and worsening ventilatory failure.

TABLE 13: Segmental innervation of the reflexes.

Reflex	*Segmental innervation*	*Normal response*
Biceps jerk	C5 (musculocutaneous nerve)	Flexion of the elbow and visible contraction of the biceps muscle
Supinator jerk	C5, C6 (radial nerve)	Contraction of the brachioradialis and flexion of the elbow and slight flexion of fingers
Triceps jerk	C6, C7 (radial nerve)	Extension of the elbow and contraction of the triceps
Knee jerk	L2, L3, L4 (femoral nerve)	Extension of the knee and visible contraction of the quadriceps
Ankle jerk	S1 (medial popliteal nerve)	Plantar flexion of the foot and visible contraction of the gastrocnemius

The diagnostic test was the anticholinesterase receptor antibody (ACRAB) that was strongly positive. She was initially treated with IV neostigmine and later with oral pyridostigmine. She responded very rapidly and did not need NIV after the first dose of neostigmine. She was later put on azathioprine. Over repeated follow-up over many months, she was weaned off the steroids, pyridostigmine, and finally the azathioprine.

SUMMARY

Examination of the neurological system in a patient with preserved consciousness is focused around a few essential questions. First, one ensures that there is no impending deterioration due to bulbar or respiratory muscle weakness. Then, one evaluates if the neurological finds are secondary to a systemic problem, septic, metabolic, or toxic. If there is a neurological disease, the evaluation should lead the clinician to the site and the etiology of the disease, that is, where is the lesion and what is the lesion.

Supplementary investigations and imaging will then be needed to guide one to confirming the diagnosis and instituting the appropriate management. Initially, and at all subsequent times, any major cardiorespiratory decompensation requires rapid evaluation and management, and this takes priority to the neurological evaluation and management.

ACKNOWLEDGMENT

The authors would like to thank Dr Charu Sankhla (Consultant Neurologist, Hinduja Hospital, Mumbai) for her valuable inputs.

SUGGESTED READING

1. Hutchison's Clinical Method, 24th edition.
2. PJ Mehta's Practical Medicine.
3. R Alagappan's Manual of Practical Medicine.

Chapter

10

Respiratory System Examination in a Critically Ill Patient

Jose Chacko

ABSTRACT

Most patients in the intensive care unit (ICU) will have respiratory system involvement upon admission or during critical illness. To approach respiratory failure in these patients, it is essential to gather comprehensive information to identify and understand the causes of respiratory failure, closely monitor the patient's condition and adjust treatments as necessary. Conducting a detailed history-taking and focused clinical examination of the patient's respiratory status and other organ dysfunctions is crucial. The chapter covers a systematic approach to examining the respiratory system of critically ill patients.

INTRODUCTION

A systematic approach to the evaluation of critically ill patients is of paramount importance. The respiratory system is commonly involved among patients who are critically ill. The key to preventing disastrous outcomes lies in the early identification of life-threatening problems with the prioritization of interventions. The clinical presentation also needs to be interfaced with the technology available at the bedside.

A stepwise approach aims to keep the patient alive with expeditious initial interventions. Once stability is attained, a more comprehensive evaluation of the history leading to the present illness and a detailed physical examination may be undertaken. The following chapter aims to provide the reader with an organized approach to the examination of the respiratory system in a critically ill patient.

CASE SCENARIO

A 72-year-old man presented to the emergency department with increasing breathlessness for the past 2 days. He started feeling unwell 5 days ago with a fever and a runny nose. He developed a dry cough a day later and began feeling easily fatigued. Two days later, the cough became productive of mildly blood-stained sputum. Initially, he felt breathless while walking to the toilet; the breathlessness gradually worsened over the next few days. He also experienced a catching type of pain over the right side of the chest while breathing in.

His past medical problems included diabetes mellitus controlled with oral hypoglycemic agents, and hypertension, for which he was on a beta-blocker. He had suffered an acute myocardial infarction 5 years ago, for which he had undergone angiography and stent placement. He is currently on dual antiplatelet therapy.

On examination, his respiratory rate was 35 breaths/min, with minimal accessory muscle use. His oxygen saturation by pulse oximetry was 92% while on supplemental oxygen, 6 L/min through a Hudson mask. He was awake and appropriate, but unable to complete sentences as he felt short of breath. His heart rate was 124 beats/min, in sinus rhythm, and his blood pressure, 115/52 (64) mm Hg. His tympanic membrane temperature was 102.4°F. Arterial blood gas analysis revealed pH: 7.52, pCO_2: 28 mm Hg, pO_2: 58 mm Hg, HCO_3: 22 mmol/L, and lactate: 2.2 mmol/L. His blood glucose level was 172 mg/dL. His peripheries were warm, with a capillary refill time of 4 seconds.

HISTORY AND SYMPTOMATOLOGY

The history of symptoms is very important with respect to patients presenting with respiratory distress. The most common respiratory symptoms at presentation to an intensive care unit (ICU) can be cough, with expectoration, breathlessness, wheezing, chest pain, or hemoptysis. Weight loss and anorexia need to be considered in patients with chronic infections such as tuberculosis

TABLE 1: Color of sputum and possible etiologies.

S. No.	Color of sputum	Possible etiologies
1.	Yellow/green	Bacterial infections
2.	Black	Pneumoconiosis
3.	Pink and frothy	Pulmonary edema
4.	Rusty sputum	Pneumococcal pneumonia
5.	Red currant jelly	*Klebsiella pneumoniae*
6.	Blood tinged	Tuberculosis
7.	Anchovy sauce	Ruptured amebic liver abscess

TABLE 2: Modified Medical Research Council (mMRC) dyspnea grading.

Grade 0	Not troubled by breathlessness except on strenuous exercise
Grade 1	Short of breath when hurrying or walking up a slight hill
Grade 2	Walks slower than contemporaries on the level because of breathlessness or has to stop for breath when walking at own pace
Grade 3	Stops for breath after walking 100 meters or after a few minutes on the level
Grade 4	Too breathless to leave the house or breathless when dressing or undressing

or malignancy. It is important to ask more details with respect to when did it start, how long, what aggravates, or relieves the symptoms, the color and quantity of sputum **(Table 1)**, and have the symptoms been static or worsening. Foul-smelling sputum indicates lung abscess, bronchiectasis, or anaerobic infections. With the onset of breathlessness within minutes, one needs to consider pneumothorax, acute pulmonary thromboembolism, pulmonary edema, or aspiration. Long duration of breathlessness should be graded based on the modified Medical Research Council (mMRC) dyspnea scale as shown in **Table 2**. The hemoptysis should be quantified as follows: mild <100 mL/day, moderate 100–150 mL/day, severe up to 200 mL/day, massive >600 mL/day or 100 mL/day for >3 days or 150 mL/h. Causes of hemoptysis include tuberculosis, malignancy, fungal infection, pulmonary thromboembolism, and vasculitis.

GENERAL PHYSICAL EXAMINATION

General examination should be rapid and focused. The physician should recognize the respiratory distress and hypoxia, assess severity, understand the etiology, and identify complications of ongoing therapy. Perform a rapid head-to-toe examination looking for specific signs. The general examination relevant to the respiratory system includes the general condition of the patient including built and nourishment. Pallor, icterus, clubbing, cyanosis, lymphadenopathy, pedal edema, tripod position, purse lip breathing, and respiratory pattern are important with respect to the respiratory system. Examination of the eye for choroid tubercules or Horner syndrome may indicate a Pancoast tumor.

SYSTEMIC EXAMINATION

The ABCs

Evaluation of the ABCs—airway, breathing, and circulation is the key to the successful resuscitation of critically ill patients. Are there signs of airway obstruction? An obstructed airway leads to noisy breathing, the gurgling of secretions, and signs of hypoxia. A simple maneuver such as head tilt or chin lift may maintain airway patency while you prepare for endotracheal intubation. An oropharyngeal or nasopharyngeal airway may be useful as a temporary intervention to overcome airway obstruction while preparing for a definitive airway. If the patient is hypoventilating or apneic, manual ventilation needs to be performed using a tight-sealing bag-valve mask device. Pulmonary aspiration may occur with this technique; hence, it is important to rapidly secure the airway by endotracheal intubation. The adequacy of ventilation is evaluated by chest wall rise and recoil. The use of an end-tidal carbon dioxide monitor attached to the manual ventilation device will also enable ascertaining the adequacy of ventilation.

Confirmation of Endotracheal Tube Position

Following intubation, a quick clinical assessment is important to check the endotracheal tube position. The chest wall may be observed to assess symmetrical expansion on both sides. Auscultation may also help confirmation of adequate ventilation on both sides. If the tube is inserted too far down, into the main bronchus on one side, the absence of ventilation on the contralateral side may be obvious. In adults, this usually results in right endobronchial intubation with absence of breath sounds on the left side. The depth of insertion of the endotracheal tube may be checked by inspecting its position at the level of the incisor teeth. The depth of insertion ranges from

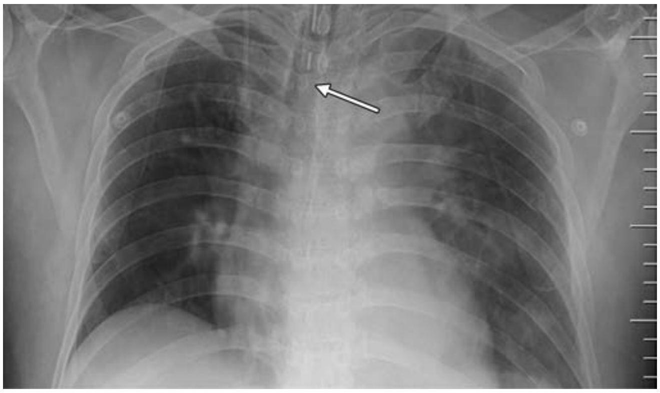


Fig. 1: Chest radiograph with the tip of the endotracheal tube seen above the carina.

20 to 22 cm in female and 21 to 23 cm in male subjects. Esophageal intubation may be difficult to diagnose by auscultation or evaluation of chest wall movement alone; however, a gurgling noise during lung insufflation and the presence of gastric contents in the endotracheal tube are tell-tale signs. Confirmation of tracheal tube placement by capnography is mandatory to avoid mishaps during airway management in critically ill patients with a low margin of safety. The position of the endotracheal tube is confirmed by bedside chest radiography **(Fig. 1)**.

EXAMINATION OF RESPIRATORY SYSTEM

The Look, Listen, and Feel Approach

Detailed examination of the respiratory system follows the "look, listen, and feel" approach **(Table 3)**.

TABLE 3: The "look, listen, and feel" approach to the examination of the respiratory system.

Look	• Does the chest expand symmetrically? Are there any chest deformities? • Are there any signs of respiratory distress? (use of accessory muscles, flaring of alae nasi, paradoxical respiration, and sweating) • Is the patient cyanotic? • Is patient-ventilator asynchrony present? • Quantity and character of secretions • Endotracheal tube position at the level of the incisor teeth
Listen	• Does the patient have stridor? • Is air entry equal on both sides? • Quality of breath sounds • Are there additional sounds? (crackles, rhonchi, wheeze, and pleural rub) • Is there any air leak around the cuff of the tracheal tube?
Feel	• Is the tracheal central? • Does the chest wall rise symmetrically? • Any local tenderness or crepitus? • Does the patient have a flail segment?

Look

The overall appearance: What position does the patient tend to assume? Most dyspneic patients are more comfortable sitting upright compared to lying supine. This phenomenon is most perceptible in heart failure. In patients with heart failure, the supine position leads to an increase in the venous return and accumulation of interstitial fluid in the lung, resulting in worsening dyspnea. Orthopnea, characterized by episodic awakening during sleep with the feeling of breathlessness, is typically experienced by patients with cardiac failure. In contrast to orthopnea, worsening of dyspnea may occur in the upright compared to the recumbent position. This phenomenon is termed platypnea and is often accompanied by hypoxia in the upright position, referred to as orthodexia. Platypnea and orthodexia are observed in patients with intracardiac right-to-left shunts, hepatopulmonary syndrome, and in pulmonary diseases with ventilation-perfusion mismatch.

Chest symmetry, respiratory excursions: There may be obvious abnormalities of the shape of the chest wall, including kyphosis, scoliosis, and pectus carinatum that may lead to restrictive lung disease. The extent and symmetry of chest wall excursions are noted. Asymmetry of chest wall excursions may be observed in unilateral lung diseases such as lobar pneumonia, collapse, pleural effusion, or pneumothorax. Multiple rib fractures with ≥2 breaks in each rib result in a "flail chest"—the segment involved gets sucked inward during spontaneous inspiratory efforts.

Respiratory rate, breathing pattern, and accessory muscle activity: The respiratory rate may be manually estimated by observing the number of chest excursions per minute. ICU monitors display the respiratory rate, algorithmically derived from the electrocardiogram (ECG) waveform. Tachypnea >30/min is usually associated with acute respiratory failure. In paradoxical respiration, the abdominal wall moves inward during inspiration. This pattern of breathing is observed in patients with airway obstruction and impairment of diaphragmatic function. Use of accessory muscles of breathing, including the sternocleidomastoids and flaring of the alae nasi, are

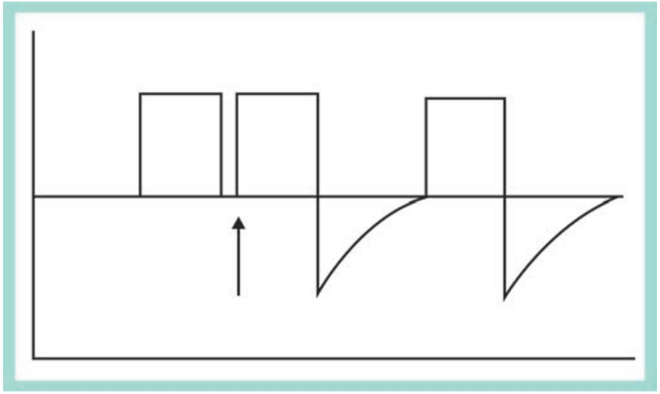


Fig. 2: Double triggering. To consecutive breaths occur following a single patient effort (arrow). Double triggering occurs when the set inspiratory time on the ventilator is shorter than the patient inspiratory time, resulting in the triggering of a second breath.

characteristic of respiratory distress with the likely requirement for support.

Patient-ventilator asynchrony: There are many different types of asynchronous breathing patterns that occur in mechanically ventilated patients. These include failure to trigger, double triggering **(Fig. 2)**, reverse triggering, and inappropriate triggering. Evidence patient-ventilator dyssynchrony is usually obvious from the ventilator display.

Inspection of secretions: In mechanically ventilated patients, the volume and character of secretions during suctioning may offer clues regarding the underlying condition. Pink, frothy secretions are typically seen in cardiogenic pulmonary edema. Occasionally, large volumes of thin, watery secretions may be seen in intubated patients due to a hypersecretory state resulting from the presence of a tracheal tube. Yellow, green, or brownish secretions usually suggest an ongoing infection. The volume of secretions may be scant in the early phase of pneumonia; however, it usually increases over time, and may be related to resolution of consolidation. Occasional aspirate of blood-stained secretions is not unusual in intubated or tracheostomized patients and may be due to minor injury to the airway mucosa. Suctioning of fresh blood from the airways is always of concern and needs to be evaluated expeditiously.

Listen

Stridor: Severe stridor, characterized by a high-pitched inspiratory noise, may be audible without a stethoscope. Minor degrees of stridor may be auscultated by placing the stethoscope over the anterior aspect of the neck. Air leak around an endotracheal or tracheostomy tube cuff is usually evident on auscultation over the trachea. Persistent gurgling noise originating from the pharynx is usually observed in patients with a poor level of sensorium who are unable to cough and clear secretions and points toward the need for a definitive airway.

Less intense or absent breath sounds: The intensity of breath sounds on auscultation may be reduced or absent due to several reasons. Patients with a thick chest wall or emphysema may have uniformly reduced intensity of breath sounds on both sides. Auscultation of the lungs is important after intubation; absence of breath sounds on one side suggests the possibility of endobronchial intubation, which may require pulling the tube back above the level of the carina. Pneumothorax with collapse of the entire lung usually leads to absence of breath sounds over the affected hemithorax. However, occasionally, breath sounds from the opposite hemithorax may be conducted across to the ipsilateral lung leading to confusion. Pleural effusions and hemothorax also result in diminished breath sounds on the affected side. Clearly, breath sounds will be less intense or absent over collapsed areas of the lung.

Rhonchi, wheeze, and crackles: Airway obstruction due to bronchospasm often results in an accessory sound during both the inspiratory and expiratory phases, referred to as rhonchi. In contrast, wheeze is heard predominantly during the expiratory phase and emanates from the narrowing of the small airways. Acute exacerbation of chronic obstructive pulmonary disease, asthma, and anaphylaxis often leads to wheezing. Crackles originate from the opening of the distal airways and alveoli during inspiration and are easily audible in pulmonary edema, consolidation, bronchitis, bronchiectasis, and interstitial lung disease. Pleural rub may often be mistaken for crackles; however, a pleural rub is localized and is heard during inspiration and expiration.

Feel

Palpation of the chest wall often offers considerable clinical information. Any asymmetry in chest wall excursions noted by inspection could be confirmed during palpation of the chest wall. Subcutaneous emphysema, which often accompanies pneumothorax, is readily palpable and easily distinguished by the characteristic crackly feel, or "crepitus." Extensive subcutaneous emphysema may appear very rapidly and is a feature of injury to the airways. Palpation of the trachea yields valuable clues regarding mediastinal shift. The trachea is deviated to the opposite side in pleural effusion and pneumothorax, while a collapsed or fibrosed lung leads to ipsilateral deviation. Tenderness over the chest wall occurs when the ribs are fractured; occasionally, a bony crepitus may be felt. A flail segment may be identified by palpation; a localized area of the chest wall is felt to move inward during inspiration.

Adjuvants to Physical Examination

In critically ill patients, the examination is extended by looking at the patient's bedspace, monitoring devices, and ventilators (noninvasive or invasive). Performing lung ultrasonography as a part of the point-of-care ultrasonography (POCUS), carefully looking at the ventilator screen for graphics, respiratory mechanics, and assessment of respiratory muscles, for example, diaphragm are of paramount importance. Lung ultrasonography helps in the diagnosis of respiratory conditions and for monitoring the progress of disease, certain maneuvers, for example, recruitment of lungs (*See* Chapters 15 and 26 for more details). Similarly, assessment of diaphragm function, looking for excursion and thickness, is frequently considered when weaning of the patients is planned.

Ventilator graphics provide crucial insights into a patient's respiratory status during mechanical ventilation. Assessment and monitoring of respiratory mechanics should be done in all mechanically ventilated patients. A careful examination of ventilator scalars, loops, and derived parameters helps us to diagnose obstructive and restrictive lung disease, changes in resistance and compliance of the respiratory system, identify overdistension, and work of breathing. Commonly assessed parameters are peak airway pressure, plateau pressure, resistance, compliance, intrinsic positive end-expiratory pressure (PEEP), dynamic hyperinflation, leaks, and parameters of weaning, etc.

Other System Examination

Examination of the cardiovascular system is performed in all patients with respiratory insufficiency. Most of the time the patient will have either coexisting cardiac problems (dilated cardiomyopathy), heart as the primary cause for respiratory compromise (pulmonary edema and respiratory failure), or simultaneous involvement of the heart during the illness (septic cardiomyopathy). Clinical examination and echocardiography are commonly used to assess the cardiac function. Though examining the jugular venous pulsations, feeling for parasternal heave, listening to S2, murmurs are performed, they are not always reliable in critically ill patients and are replaced by bedside echocardiography. *See* Chapter 26 for details.

Venous ultrasound is an extension for examining a patent with cardiorespiratory compromise especially with suspicion of pulmonary embolism. A quick three-point deep vein screening of the femoral and popliteal veins is helpful.

A focused central nervous examination may be needed in case of hypoventilation, diaphragmatic paralysis and in case of quadriplegia. *See* Chapters 8, 9, and 20 for more details.

Follow-up of the Patient

Our patient was tachypneic and was unable to complete full sentences. His oxygen saturation was low (92%) with supplemental oxygen, and his arterial blood gas analysis confirmed a wide A-a gradient. Assuming an FiO_2 of 0.4 with oxygen 6 L/min through a Hudson mask, given a PaO_2 of 58 mm Hg, his PaO_2/FiO_2 ratio was 145 mm Hg. On inspection, his chest wall excursions appeared to be less on the right side. Auscultation confirmed reduced breath sounds over the right infraclavicular and axillary areas with scattered crackles. Ultrasonography of the lung revealed consolidation of the right upper zone **(Fig. 3)**, confirmed on the chest radiograph. He was commenced on intravenous antibiotics. As he was being evaluated in the emergency department, his dyspnea progressively

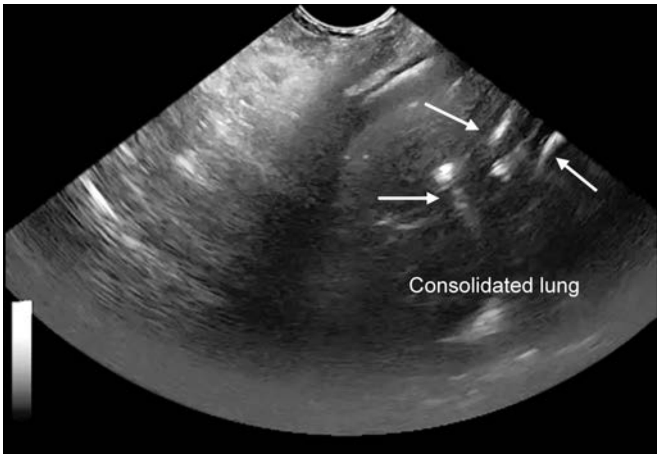


Fig. 3: Ultrasonography of the lung showing consolidation. Air bronchograms (arrows) are seen against the background of a "tissue-like" lung.

worsened, with a drop in the oxygen saturation to <90%. Hence, he was commenced on noninvasive ventilatory support with an inspiratory positive airway pressure (IPAP) of 10 and expiratory positive airway pressure (EPAP) of 5 cmH_2O and was transferred to the ICU.

Course in the Intensive Care Unit

Our patient continued to worsen, with increasing tachypnea, with a further drop in oxygen saturation down to 80–85%. When you see him a few hours after, he presented to the emergency department, he was in obvious distress, trying to pull out the noninvasive ventilation (NIV) mask. His blood pressure read 118/48 (71) mm Hg with a heart rate of 132 beats/min. You intubated him following intravenous fentanyl 200 µg, midazolam 2 mg, and rocuronium 80 mg. Endotracheal tube placement was confirmed by capnography. On auscultation, he continued to have reduced breath sounds over the right upper lung zones. His initial ventilator settings on the volume-control assist control mode were as follows: FiO_2 1.0, tidal volume: 400 mL, respiratory rate: 20 breaths/min, I:E ratio 1:2, and PEEP: 5 cmH_2O. His oxygen saturation was maintained >95% on an FiO_2 of 0.5. Following intubation, he developed hypotension with a mean arterial pressure (MAP) of <60 mm Hg. You resuscitated him with 1.0 L of Ringer lactate over 1 hour. A norepinephrine infusion was commenced to maintain the MAP >70 mm Hg. As the MAP remained low, you continued to administer boluses of Ringer lactate, adding up to 3 L over the next few hours.

Worsening Hypoxia

Over the next half an hour, the oxygen saturation dropped; you increased the FiO_2 to 0.8. The MAP remained above 70 mm Hg on a stable dose of norepinephrine. You auscultated him again and heard extensive crackles on both sides, in contrast to the initial localized findings on the right upper zones. A repeat chest radiograph at this stage revealed bilateral extensive infiltrates **(Fig. 4)**, in contrast to the localized findings on the initial image. What could have resulted in bilateral involvement on clinical examination and radiography? The likely possibilities may be, first, the natural progression of the disease, with pneumonia and sepsis leading to acute respiratory distress syndrome. Second, fluid resuscitation, combined with sepsis-induced capillary leak, may have resulted in pulmonary edema. Third, the left ventricular function may be impaired, contributing to fluid accumulation in the lungs. You performed bedside echocardiography that confirmed reduced left ventricular systolic function with an ejection fraction of <40%. Considering excessive fluid accumulation in the lung and worsening hypoxia, you increased the PEEP level to 10 cmH_2O. The increase in PEEP resulted in a gradual improvement of oxygen saturation enabling a reduction in the FiO_2 to 0.4. An arterial blood gas analysis at this stage revealed pH: 7.38, pCO_2: 34 mm Hg, pO_2: 78 mm Hg, HCO_3: 23 mmol/L, and lactate: 1.8 mmol/L.

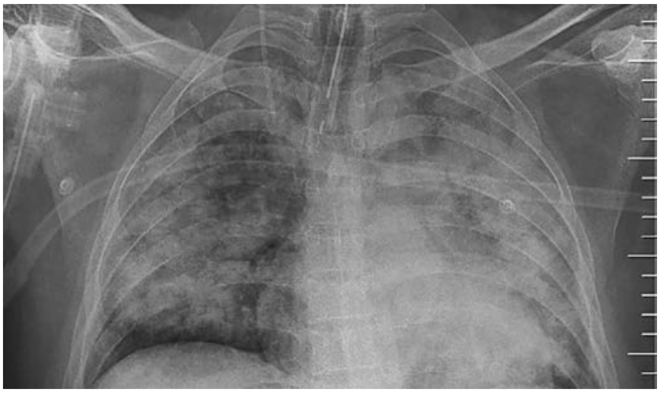


Fig. 4: Bilateral extensive infiltrates suggestive of acute respiratory distress syndrome.

SUMMARY

Critically ill patients with respiratory illness are at a high risk of deterioration, and their clinical condition needs to be closely monitored. A systematic clinical examination in conjunction with pulse oximetry, bedside ultrasonography, and chest radiography usually enables rapid evaluation of a worsening situation and timely action. It is important to rapidly assess the ABCs in critically ill patients. If airway compromise is present, expeditious action is called for, usually with endotracheal intubation. The systematic approach involves three key elements—look, listen, and feel. In the case discussed above, there was a strong suspicion of consolidation considering the history and clinical examination, corroborated by the findings on bedside ultrasonography and chest radiography. During the initial course of resuscitation, he developed worsening oxygenation; the clinical findings pointed toward fluid accumulation in the lungs and were confirmed on imaging. A combined approach including clinical evaluation and imaging enabled rapid evaluation of his condition, followed by interventions, including an increase in the level of PEEP that resulted in improved oxygenation.

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Chapter

11

Cardiovascular System Examination in a Critically Ill Patient

Ram E Rajagopalan

ABSTRACT

Given the instability of a critically ill patient with cardiovascular disease, clinical evaluation by traditional assessment methods may be constrained by the available time. It is therefore common to obtain an abbreviated clinical history and to perform a focused clinical examination to arrive at a working diagnosis while the patient is being stabilized. This evaluation not only involves physical examination of the central and peripheral circulation but also utilizes feedback from initial therapeutic support maneuvers and from a wide variety of point-of-care instruments that help narrow down the differential diagnosis. Often a reasonable working diagnosis is established by this process. Subsequent assessment should aim at fine-tuning the diagnosis using established techniques and tools. This delayed evaluation should be more comprehensive and should also focus on systematic evaluations of other organ systems that may be impacted by the cardiovascular problem.

INTRODUCTION

Evaluation of a critically ill patient is often a challenge, as it is confounded by limitations in our ability to perform a thorough physical examination in an unstable patient with a rapidly evolving, life-threatening clinical disorder. As a result, a quick and focused physical evaluation is often used in an emergency, whereas a broader detailed evaluation can be performed after patient stabilization. The relative inefficiency of standard physical diagnostic methodology makes us rely on a variety of technological tools to assess such a seriously ill individual within the tightly restricted time frame that is often available to us. The success of such an assessment will depend on the integration of traditional physical diagnostic inputs with the information obtained from bedside technology. The evaluation of the cardiovascular system will often involve the use of inputs from electrocardiographic and hemodynamic monitoring, as well as from point-of-care (POC) echocardiographic evaluation.

This will be illustrated in the case discussed further.

THE RAPID, FOCUSED INITIAL EVALUATION

History

A 48-year-old man with no prior known comorbidity presents with a 1-day history of severe midsternal chest discomfort and dyspnea. He was apparently normal other than feeling "feverish" and having mild upper respiratory symptoms over the last 3–4 days, that was not very different from prior "colds."

A quick elaboration of the history is needed, and the patient says that his symptoms are a midsternal pain that came on spontaneously at rest and dyspnea of increasing severity. The pain was initially mild and noted about 2–3 days ago, but has been more intense and increasing in severity in the last hour before seeking advice. There is radiation of the pain to the jaws and arms. The pain may have a postural variation, severe in any position but with marginal relief while seated. The dyspnea, likewise, has been gradually increasing in intensity over a similar time frame and is present currently even at rest. There is a mild cough but no productive sputum.

Examination

The working hypothesis at this time points to a primary cardiac or respiratory diagnosis. The preliminary approach would be to assess the patient's clinical stability and to provide supportive care, if needed. A more detailed history and physical evaluation may be delayed until stabilization. One approach is to the use of the older Advanced Cardiovascular Life Support (ACLS) "triads" **(Box 1)** for this initial process.

BOX 1: Initial assessment and support will have to proceed in parallel. The Advanced Cardiovascular Life Support (ACLS) suggested triads are a good way to guarantee stabilization of the patient while the initial clinical inputs indicate the diagnostic direction.

Assessment triads (Modified from ACLS-based recommendations):

1. *Critical ABCs:* Airway–Breathing–Circulation
2. *Vital signs:* Pulse rate–blood pressure (BP)–Respiratory rate
3. *Stabilization:* Intravenous (IV)–Oxygen–Monitor [electrocardiogram (ECG)/oximetry]
4. *Etiology:* Rate/rhythm–Volume–Cardiac function

The absence of a cardiorespiratory arrest is obvious and implies a preservation of the *airway–breathing–circulation* (i.e., the ABCs) triad. If these were compromised, the patient might have needed cardiopulmonary resuscitation and mechanical support of breathing.

This shifts the focus to the next triad of *pulse rate–blood pressure (BP)–respiratory rate*, the "vital signs" of cardiorespiratory function. Palpation of the pulse at large arterial sites (carotid and femoral) is preferred over peripheral sites (radial), where vasoconstriction may make the assessment difficult. A palpable pulse will reassure the physician of the presence, if not the adequacy, of circulation and allow the detection of irregular rhythms, tachy- and bradyarrhythmia that may be compromising the circulation. A noninvasive BP (sphygmomanometry or preferably an automated oscillometric device) is essential at this time to detect the stability of the patient. The respiratory rate can also be observed in parallel.

In this patient, the pulse was palpable in major arteries, but was attenuated in the peripheral sites. The heart rate was about 110–120 beats/min and the BP was 90/84 mm Hg. His respiratory rate was about 24 breaths/min, with a respiratory paradox (abdominothoracic asynchrony), but with no accessory muscle use or intercostal indrawing.

With signs of compromised cardiorespiratory function, the next triad of actions is to obtain an intravenous *(IV) access*—provide *oxygen*—and use suitable *monitoring*. The IV access at this time provides a route for subsequent therapy with medication or fluids as needed and for the withdrawal of blood for laboratory sampling. Given the routine availability of pulse oximetry, oxygen therapy in this dyspneic patient can be based on the saturation obtained by pulse oximetry. If there is significant vasoconstriction, the pulse oximetric tracing may be absent and empirical application of high oxygen concentrations (e.g., through a nonrebreather reservoir bag mask) may be appropriate.

BOX 2: Standardized measurement of finger-bed capillary refill identifies hypoperfusion (not just hypovolemia). The use of chronometry to assess the refill time may reduce interobserver variability.

Methodology: Capillary refill time (CRT)

- Apply firm pressure to the ventral surface of the distal phalanx of the index finger with a glass slide
- The pressure is increased until the skin blanches and maintained for 10 seconds
- Assess the time for return of the normal skin color preferably with a chronometer/stopwatch
  - A CRT >3 s is abnormal

Source: Hernández G, Ospina-Tascón GA, Damiani LP, Estenssoro E, Dubin A, Hurtado J, et al. Effect of a resuscitation strategy targeting peripheral perfusion status vs serum lactate levels on 28-day mortality among patients with septic shock; The ANDROMEDA-SHOCK Randomized Clinical Trial. JAMA. 2019;321(7):654-64.

Simultaneously, other monitors, mainly a single-channel electrocardiogram (ECG) and noninvasive automated BP measurement, are appropriate.

The ECG monitor shows a sinus rhythm with a heart rate near 120 beats/min. Oximetric saturation on room air is 98–100%. Considering the dyspnea, the patient is anyway placed on oxygen delivered as 2 L of flow by nasal prongs. The BP on repeated measurement hovers at systolic pressure of 90–95 mm Hg and about 80–85 mm Hg, diastolic.

With the recognition that the patient's condition is compromised, but stable, a more detailed focused evaluation of the cardiovascular system is appropriate at this point. Once again, the triad-based approach would allow us to quickly differentiate the cause of the underlying hemodynamic problem. Is it *rate/rhythm-related–volume status-related–or cardiac functional compromise?*

While the single-channel ECG is useful to identify the contribution of heart rate (very high or low) or arrhythmia to the clinical state, it is less likely in this case, with mild sinus tachycardia, to be of significance. A focused evaluation of the cardiovascular function at this time would allow a better differentiation of filling (volume) problems from cardiac function issues. This could be done using several modes of traditional physical examination.

- *Inspection of the peripheral circulation:* Commonly, we look for gross signs of peripheral underperfusion such as cyanosis or symmetrical peripheral vasoconstriction as markers of impaired cardiac function. Although a careful inspection of the peripheries for skin changes, mottling, cyanosis, and edema are useful, a systematic assessment of *capillary refill times* (CRT, **Box 2**) and

BOX 3: The hepatojugular (abdominojugular) reflux is a fair indicator of the right heart failure, but may also be diagnostic of congestive failure in tachypneic patients. A standardized method of assessment is described.

Methodology: Hepatojugular reflux
- With the patient in a semirecumbent position (head elevation 45°), observe jugular pulsation during quiet respirations [baseline jugular venous pressure (JVP)]
- Apply gentle pressure (30–40 mm Hg) over the right upper quadrant or mid-abdomen for 15 s
- Recheck the JVP

A rise of JVP >3 cm is a positive test.

In normal subjects, JVP may decrease (due to reduced venous return) or may transiently rise and then return to normal or decrease within 10 s

Source: Wiese J. The abdominojugular reflux sign. Am J Med. 2000;109(1):59-61.

the *hepatojugular reflux* (H-JR, **Box 3**) may contribute significantly to narrowing the differential diagnosis. Standardization of the methodology for CRT **(Box 2)** has allowed it to be used as a titratable indicator of low perfusion. Though the H-JR is seen in a variety of conditions characterized by high venous pressure and low right ventricular (RV) compliance (including RV failure, pulmonary hypertension, constrictive pericarditis, and restrictive cardiomyopathy), it has very high specificity in identifying left heart failure and elevated pulmonary artery occlusion pressure in tachypneic patients and is never seen in pericardial tamponade.

- *Palpation of peripheral pulses:* Evaluation of the pulse in major (carotid and femoral) and peripheral (radial, brachial, and dorsalis pedis) arteries is part of the routine evaluation. Palpability only in the major vessels either implies peripheral vascular disease or, if symmetrical, a low perfusion state. The volume of the pulse is low if cardiac output is compromised and differentiates it from sepsis and aortic regurgitation, which have high volume pulses. Irregularities caused by arrhythmia may be detected.
- *Auscultation of cardiac sounds:* Often, cardiac auscultation may be difficult in the critically ill patient with cardiorespiratory disorders, either because of the noisy environment or more often due to adventitious respiratory sounds from pulmonary congestion or lower respiratory secretions. However, a careful auscultation to detect murmurs and rubs could be of value.
- *Auscultation of respiratory sounds:* Listening for the fine rales characteristic of pulmonary edema and differentiating them from the coarser sounds of lower respiratory pathology is useful. Rhonchi, though characteristic of airway narrowing, may often be a feature of cardiogenic pulmonary edema.

In our patient, there are palpably cold extremities, but with no discoloration, no cyanosis, skin changes, or edema. The peripheral pulses, though well felt in major arteries, are more difficult to palpate in the radial and dorsalis pedis arteries on both sides. The pulses are symmetrical bilaterally and are of low volume. The CRT is significantly prolonged (about 10 s). The jugular venous pressure (JVP) is about 8 cm (elevated) above the angle of Louis with a 30° elevation of the head of the bed, but there is no hepatojugular reflux seen. Cardiac sounds are muffled and difficult to hear, but there seems to be an evasive high-pitched murmur or rub in the left parasternal area. The lung fields are normal, with clear breath sounds and no adventitious sounds on either side.

This rapid evaluation helps to narrow our differential diagnosis considerably. In this hypotensive patient, the symmetrically cold distal extremities imply an elevated systemic resistance, eliminating concerns about a vasodilated septic state. This is supported by the low volume pulse and the narrow pulse pressure (systolic minus diastolic blood pressure) on repeated evaluation. The prolonged CRT supports the existence of a low-perfusion state. The H-JR, though a very specific indicator of volume overload, is not sufficiently sensitive to identify congestive failure. Thus, while a positive response virtually confirms volume overload in a tachypneic patient, the absence of a response may be a false negative result or may imply conditions such as pericardial tamponade. Lung congestion would be seen with the volume overloaded states and the clear lung fields on auscultation divert the diagnosis toward acute right heart problems (pulmonary embolism or RV infarction) or pericardial tamponade. The muffled heart sounds and questionable pericardial rub push us toward the latter.

At this stage, it is important to realize that some kind of therapeutic maneuver may often be required to stabilize the patient (e.g., fluid boluses if the patient is suspected to be hypovolemic) and feedback from the patient's response to such treatment would confirm or alter the diagnostic direction significantly. The use of POC diagnostic and monitoring technology becomes a useful adjunctive tool at this stage **(Box 4)**.

The 12-lead ECG is ideal to confirm the existence myocardial ischemia or infarction and to identify arrhythmias and bundle branch blocks. A multitude of cardiopulmonary conditions, including pulmonary emboli, pericarditis, myocarditis, and electrolyte imbalance will also be identifiable.

Laboratory tests especially high troponin levels will point toward myocardial damage (ischemia or myocarditis), but may also be elevated in pulmonary embolism and extracardiac conditions especially in patients with renal dysfunction. Elevations of natriuretic peptide levels [brain natriuretic peptide (BNP), N-Terminal (NT) pro BNP] imply congestive heart failure or volume overload due to renal dysfunction. Right heart dilatation from pulmonary hypertension and pulmonary emboli will also cause elevations. POC measurement technology facilitates their use for immediate diagnostic decisions. Detection of hypoxia, metabolic or respiratory acidosis on the *arterial blood gas analysis* would also aid diagnosis.

BOX 4: Point-of-care (POC) tools that can aid in the identification of the etiology of cardiopulmonary compromise.

POC cardiac assessment:
- 12-lead electrocardiogram
- *POC laboratory tests:* Troponin and natriuretic peptides
- Arterial blood gas analysis
- User-directed echocardiography
- Invasive arterial pressure monitoring

Point-of-care ultrasonography performed by the intensivist would contribute greatly to the differential diagnosis of the cardiac patient. Though it is not performed at the expertise level of a trained cardiologist, it can allow a rapid identification of the primary mechanisms responsible for the decompensation of the patient **(Fig. 1)**. It is not restricted to echocardiographic evaluation, but is often combined with sonographic evaluation of the lung as well, allowing a differentiation of cardiogenic pulmonary oedema from pulmonary injury **(Fig. 2)**.

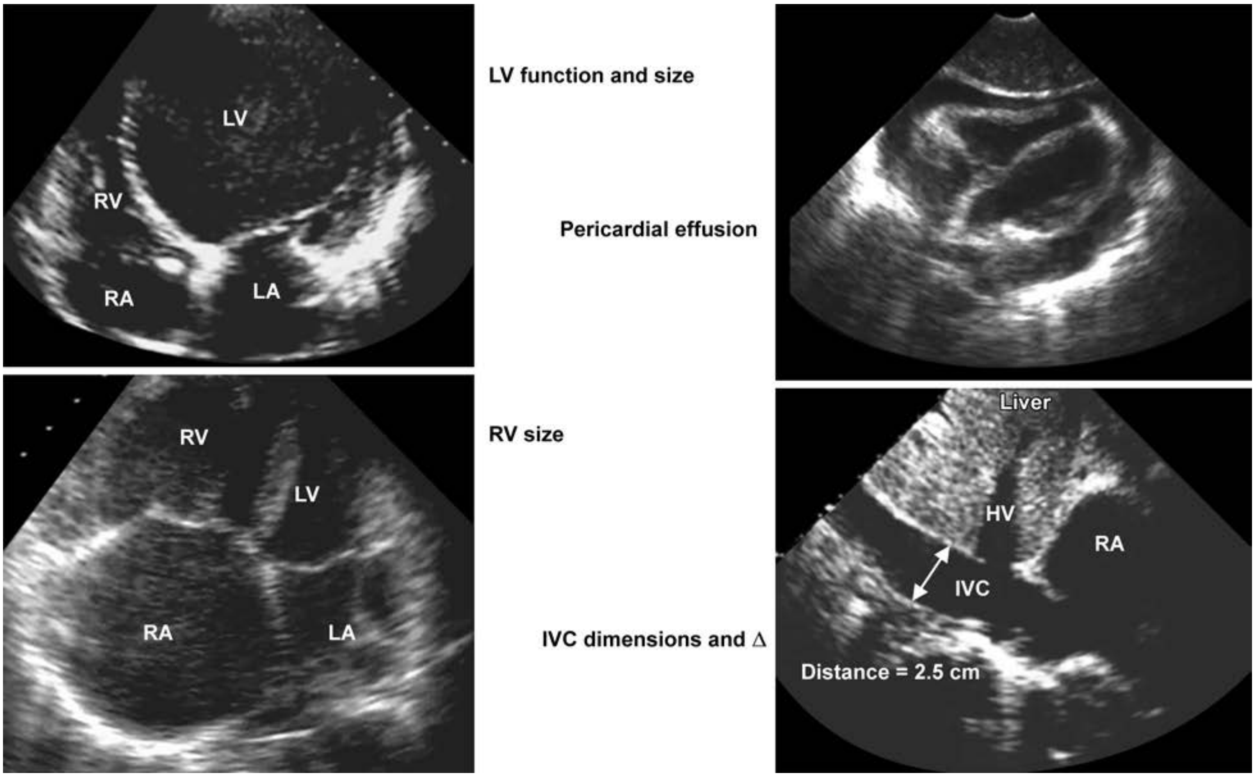


Fig. 1: Point-of-care ultrasonography performed by a "Level I" (novice) evaluator will often be adequate in identifying the cause of the underlying cardiovascular compromise. Qualitative evaluation of the left ventricle (LV) size and function, right ventricle (RV) dimension, the existence of pericardial effusion, and the filling of the inferior vena cava (IVC; to identify volume loading) will provide the basic diagnostic direction. (HV: hepatic vein; LA: left atrial; RA: right atrial)

Invasive arterial pressures may usually be placed at this time. Besides giving real-time assessment of hemodynamic adequacy, it can be a valuable tool for the differentiation of the etiological origin of the cardiac problem.

In this patient, the ECG **(Fig. 3)** *shows diffuse ST elevation (except aVR and V1) without reciprocal changes. Ischemia is less likely than pericarditis. The clear-cut PR depression and the down-sloping TP segment (Spodick sign) (lead II) add more validity to the latter diagnosis.*

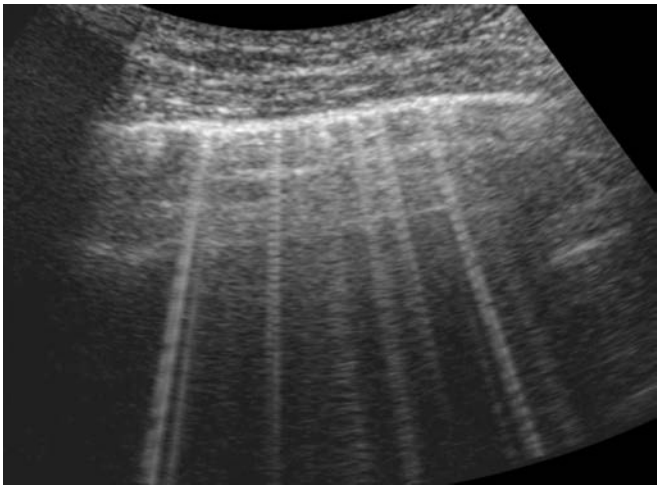


Fig. 2: On the lung ultrasound, "septal B-lines" have near perfect sensitivity and specificity for identifying cardiogenic pulmonary edema.

Note: Discrete vertical hyperechoic artifacts with a narrow base at pleural line, extending through the screen without fading and moving synchronously with lung sliding.

Source: Soldati G, Demi M, Demi L. Ultrasound patterns in pulmonary edema. Ann Transl Med. 2019;7(Suppl 1):S16.

There are no elevations of the troponins or BNP. The arterial blood gas is compatible with a combined metabolic acidosis and respiratory alkalosis. The serum lactate is elevated to 4 mmol/L, but the oxygenation is adequate (pO_2 *is 98 mm Hg on room air).*

The POC 2D echocardiogram (2DE) **(Fig. 4)** *shows a large pericardial effusion. Left and right ventricular dimensions and function appear normal. There is right atrial collapse, but no ventricular collapse on the 2DE. The inferior cava is distended (2 cm diameter) and does not collapse with spontaneous breathing. The lung ultrasound shows no pathology.*

An arterial cannula placed in the right radial artery shows significant decline of systolic pressure (over 15 mm Hg) during normal inspiration, compatible with a pulsus paradoxus **(Fig. 5)**.

It is clear from the development of the case that, while early directed clinical evaluation narrows the diagnosis, it is seldom definitive. Appropriate addition of bedside technology is of great value at this stage. In this case, the 12-lead ECG directs the clinician to consider the relatively less common diagnosis of pericardial disease, which the clinical examination was implying.

The POC ultrasound directs the diagnosis further in this direction by identifying a large pericardial effusion. Unfortunately, the skills possessed by the average physician using POC ultrasound are inadequate to identify if the effusion is causing tamponade and compromising

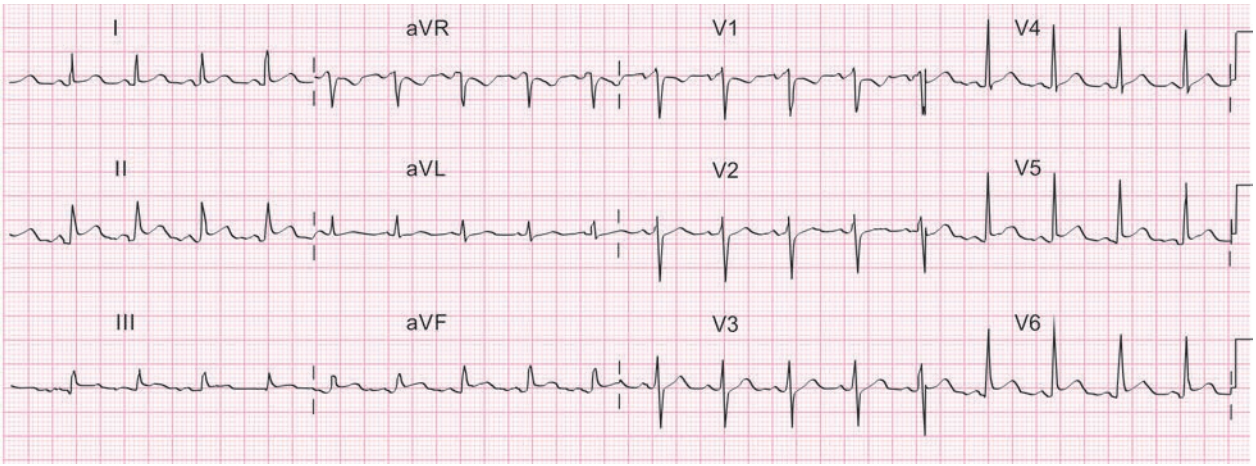


Fig. 3: A 12-lead electrocardiogram (ECG) is an essential part of the initial point-of-care (POC) evaluation of the cardiovascular system. In this case, the diffuse ST elevation and characteristics compatible with pericarditis (see text) clinch the diagnosis.

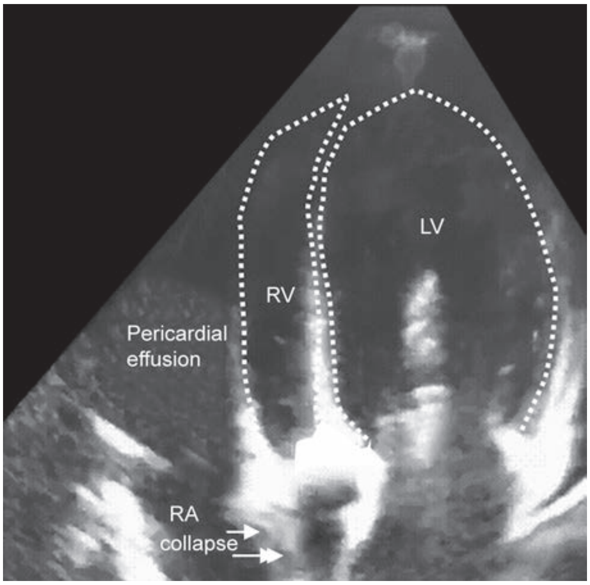


Fig. 4: The large pericardial effusion in this hemodynamically compromised patient is indicative of the cause. Right atrial (RA) collapse on the level I echocardiogram is suggestive of pericardial tamponade and will require confirmation by expert evaluation of other echocardiographic features or hemodynamic assessment. (LV: left ventricle; RV: right ventricle)

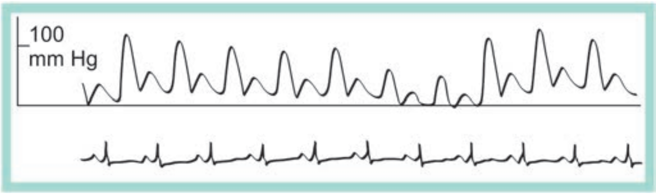


Fig. 5: Invasive arterial pressure measurement not only allows close monitoring of the compromised hemodynamic state but may also give subtle evidence of the underlying etiology. In this case, the variation in systolic blood pressure with spontaneous breathing identifies pulsus paradoxus compatible with pericardial tamponade.

cardiac function. This often requires higher skills to identify ventricular chamber collapse and flow reversal in the hepatic vein. The limitations to user-directed POC ultrasound should be recognized.

However, the identification of pulsus paradoxus on the invasive arterial line is more conclusive. It may be argued that a careful clinical assessment of respiratory variation in the systolic pressure may have been elicited by routine sphygmomanometry, without needing invasive measurement. However, a physician performing an urgent patient evaluation has limited time to focus on such subtleties. An easily accessible invasive tool improves our efficiency in detecting such unusual findings.

Thus, a rapid, focused cardiac evaluation incorporating both physical signs and technological inputs allows a rapid identification of the problem. Recently, hemodynamic profiles have been developed using these initial parameters, for instance, to guide a time-sensitive and individualized approach to initial care of the patient with heart failure **(Figs. 6A and B)**.

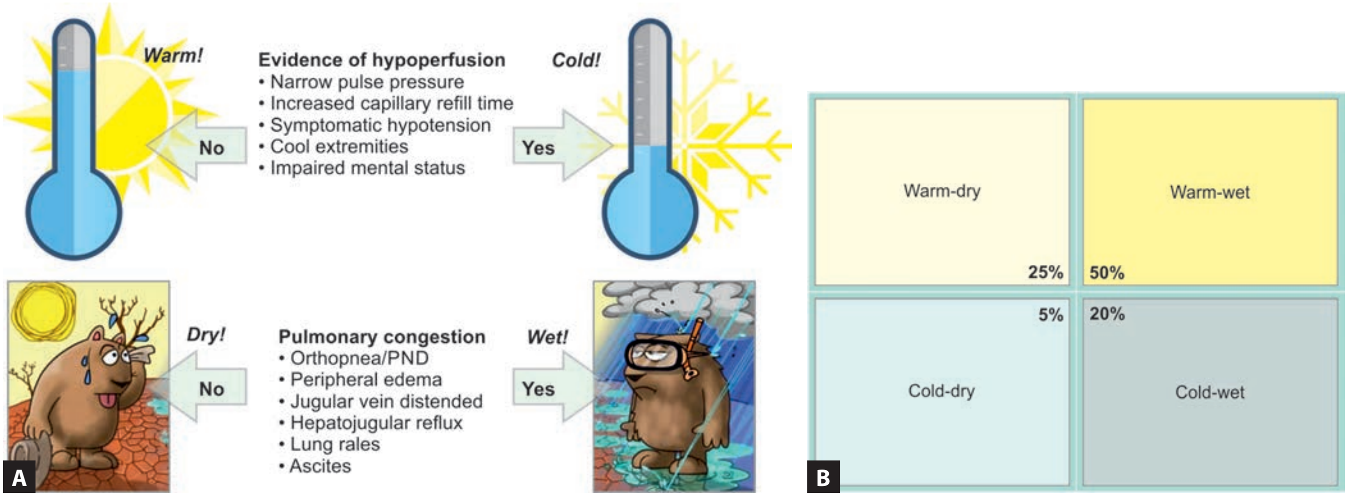
Flowchart 1 provides a summary of this approach to clinical evaluation of the cardiovascular system in the critically ill patient. Further assessment of the patient and follow-up while in the intensive care unit should be a planned, broad-based examination of other physiological systems in a comprehensive manner.

PLANNED AND DETAILED EXAMINATION

After the initial stabilization and, when possible, the identification of the primary issue that will direct the clinical care, the physician would have time to perform a more detailed evaluation and to obtain a comprehensive history to strengthen the diagnosis and to assess the progression of the patient.

Clinical evaluation of the cardiovascular patient in the intensive care unit now becomes a structured process in which all major organ systems are assessed systematically. While the main focus should remain on the patient's primary clinical presentation, a structured methodology in reviewing all major organ systems will minimize the overlooking of important supplementary information. This contrasts distinctly from the routine clinical evaluation in general medical practice, where the targeted organ alone is assessed in detail while a broader "general examination" is used for assessing other organ systems. While detailed discussion of such evaluation may be available in other chapters, **Table 1** provides an outline of the approach.

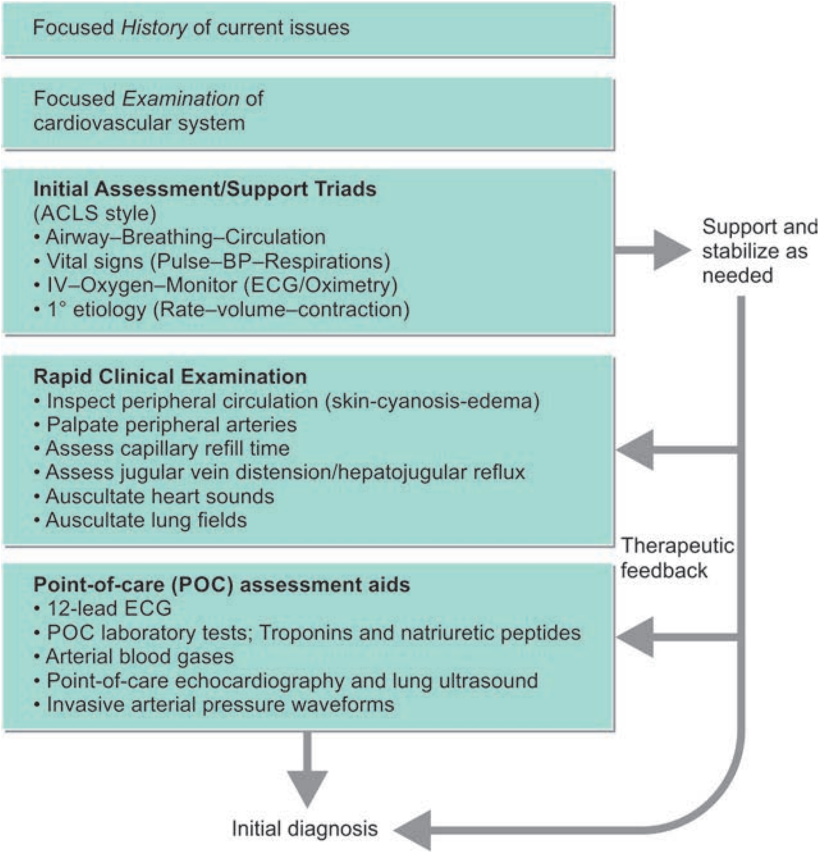
In our patient, the impaired perfusion may lead to neurological compromise and a careful sequential evaluation is required. Focal deficits, though unlikely, should also be monitored. The respiratory compromise also needs attention and he should be observed closely for potential airway compromise. Renal function may become impaired and the patient should be evaluated repeatedly for volume and metabolic complications related to this as well.


Warm!
Evidence of hypoperfusion
• Narrow pulse pressure
• Increased capillary refill time
• Symptomatic hypotension
• Cool extremities
• Impaired mental status
Cold!
No
Yes
Dry!
Pulmonary congestion
• Orthopnea/PND
• Peripheral edema
• Jugular vein distended
• Hepatojugular reflux
• Lung rales
• Ascites
Wet!
No
Yes
A
Warm-dry
25%
Warm-wet
50%
5%
Cold-dry
20%
Cold-wet
B

Figs. 6A and B: (A) Clinical findings and history used to identify "hemodynamic profiles" in patients with heart failure. Perfusion state is identified as being adequate (warm) or impaired (cold) and is combined with congestive features (wet or dry) to identify four profiles (B) that may be approached by customized therapeutic options. (PND: paroxysmal nocturnal dyspnea)

Source: Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Eur Heart J. 2016;37(27):2129-200.

Flowchart 1: Focused approach to acute evaluation of the cardiovascular system in the critically ill patient.


Focused History of current issues
Focused Examination of cardiovascular system
Initial Assessment/Support Triads (ACLS style)
• Airway–Breathing–Circulation
• Vital signs (Pulse–BP–Respirations)
• IV–Oxygen–Monitor (ECG/Oximetry)
• 1° etiology (Rate–volume–contraction)
Support and stabilize as needed
Rapid Clinical Examination
• Inspect peripheral circulation (skin-cyanosis-edema)
• Palpate peripheral arteries
• Assess capillary refill time
• Assess jugular vein distension/hepatojugular reflux
• Auscultate heart sounds
• Auscultate lung fields
Therapeutic feedback
Point-of-care (POC) assessment aids
• 12-lead ECG
• POC laboratory tests; Troponins and natriuretic peptides
• Arterial blood gases
• Point-of-care echocardiography and lung ultrasound
• Invasive arterial pressure waveforms
Initial diagnosis

(BP: blood pressure; ECG: electrocardiogram)

TABLE 1: Summary of a systematic and comprehensive approach to general evaluation of the cardiovascular patient after initial stabilization (planned, detailed evaluation).

Organ system	***1° variable***	***Related 2° variable***
General	• Overall appearance • Acute distress • Level of consciousness	Sedation and analgesia
CNS	• Consciousness/GCS • Pain level • CAM-ICU • General and focal neurological function	• Sedation • Analgesia
Respiratory	• Physical examination • Oximetric saturation • ABG • Ventilator settings • POC imaging (Lung USG/X-ray/CT)	• Distress (tachypnea, respiratory paradox, nasal flaring, intercostal retraction, etc.) • Breath sounds/adventitious sounds • FiO_2, PEEP, tidal volume, peak, plateau and driving pressure • Chest tubes
Abdomen	• Physical examination • GI function • Nutrition • POC imaging (USG abdomen)	• Bowel sounds; distension/girth, tenderness • NG tube output; bowel movements • Enteral or parenteral support • Free fluid; gallbladder
Renal	• Fluid balance • Urine output • POC imaging (Renal USG)	• Edema • Bladder distension • Hydronephrosis; renal and perinephric pathology
Skin	Physical examination	Rashes, edema, decubiti, infections, cyanosis
Catheters/tubes/drains	• Inspect and palpate vascular access sites • Foley catheter • Surgical drains and catheters	• Tenderness, erythema, purulence • Urinary turbidity, purulence, hemorrhage • Wound dehiscence, erythema, purulence of site and drain fluid
Laboratory tests/ investigations	–	
Medication	–	

(ABG: arterial blood gas; CAM-ICU: confusion assessment method in ICU; CNS: central nervous system; CT: computed tomography; GCS: Glasgow Coma Scale; GI: gastrointestinal; NG: nasogastric; PEEP: positive end-expiratory pressure; POC: point-of-care; USG: ultrasonography)

Source: Adapted from Illuzzi E, Gillespie M. Physical Examination in the ICU. In: Oropello JM, Pastores SM, Kvetan V (Eds). Critical Care [Internet]. New York, NY: McGraw-Hill Education; 2017.

SUMMARY

The approach to clinical evaluation of the cardiovascular system in a critically ill patient is strongly influenced by the clinical status and stability of the individual. In the initially unstable stage, the ability to "think on one's feet" and to seamlessly integrate basic clinical evaluation with inputs from treatment responses and POC technology is highly essential. This is summarized in **Flowchart 1**. Subsequent evaluation is focused on observation of the progression of the cardiovascular compromise, while at the same time using clinical evaluation and bedside technology to fine tune the diagnosis and therapy. Involvement of other organ dysfunction is an essential component of these planned assessments as well and should be systematically executed with attention to detail.

SUGGESTED READING

1. Hernández G, Ospina-Tascón GA, Damiani LP, Estenssoro E, Dubin A, Hurtado J, et al. Effect of a resuscitation strategy targeting peripheral perfusion status vs serum lactate levels on 28-day mortality among patients with septic shock; The ANDROMEDA-SHOCK Randomized Clinical Trial. JAMA. 2019;321(7):654-64.
2. Illuzzi E, Gillespie M. Physical Examination in the ICU. In: Oropello JM, Pastores SM, Kvetan V (Eds). Critical Care [Internet]. New York, NY: McGraw-Hill Education; 2017.
3. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Eur Heart J. 2016;37(27):2129-200.
4. Wiese J. The abdominojugular reflux sign. Am J Med. 2000;109(1):59-61.

Chapter

12

Gastrointestinal System Examination in a Critically Ill Patient

Raghavendra Kotal, Pradeep Rangappa

ABSTRACT

Abdominal examination in critically ill patients ensues its own challenges due to numerous etiologies posing diagnostic dilemma. Evaluation of abdominal pathology in intensive care unit (ICU) patients poses challenge in precise history-taking, sequential physical examination and analyzing radiological and laboratory investigations. Moreover, evidence shows that delay in the evaluation of such patients increases morbidity and mortality. However, following a methodical and in-depth process of clinical examination of the abdomen can help in identifying the pathology in these patients.

INTRODUCTION

Primary gastrointestinal (GI) disorders are one of the major reasons for intensive care unit (ICU) admission; moreover, issues pertaining to any critically ill conditions such as hypoperfusion, hypoxia, and systemic inflammation can trigger GI injuries in admitted ICU patients. Abdominal conditions encountered in the ICU range from infection, hemorrhage, trauma, ischemia, perforation, obstruction, distension, dysmotility, and inflammatory pathologies. Furthermore, intra-abdominal disorders, specifically abdominal sepsis, are associated with high mortality of up to 30% as established by a study from Abdominal Sepsis Study (AbSeS) group of the European Society of Intensive Care Medicine (ESICM).

The basic structure of assessment is history-taking, physical examination, analyzing radiological and laboratory investigations, and interventional modalities for evaluation. The difficulty in assessing critically ill starts from unreliable history-taking process as patients can be drowsy, disoriented, sedated, intubated, and mechanically ventilated. Besides, there can be diversion of attention due to encountered hemodynamic instability, respiratory distress, and other distracting injuries, which deserve immediate attention and stabilization. In fact, physical examination has a sensitivity of 82% and specificity of just 45% in blunt trauma abdomen and in abdominal abscess, it is found to be beneficial in only 43–69% of cases. Additionally, the diagnostic dilemma of suspected abdominal pathologies is due to innumerable etiologies, which can delay or mislead diagnosis, hence further increasing morbidity and mortality.

CLINICAL SCENARIO

A 54-year-old male patient with background of diabetes mellitus and hypertension presented with complaints of pain abdomen for 1 day. On arrival, he was having a pulse rate of 112 beats/min and blood pressure of 86/54 mm Hg. On eliciting history, patient pointed complaints of dull aching pain on the right upper quadrant (RUQ) region radiating to the right shoulder and severity of pain as accessed with numerical rating score (NRS) was 5/10. On inspection of the abdomen, its shape and contour were rounded or protuberant (obese) indicating his nutritional status and all quadrants moved equally with respiration. On palpation, murphy's sign was positive with tenderness noted on the RUQ region. Auscultation for the bowel movements showed normal bowel movements with no bruits and percussion of the abdomen was tympanic. After initial stabilization with intravenous fluids, antibiotics and basic blood investigations were noted. He underwent contrast-enhanced computer tomography, which showed distended gall bladder with focal irregularities in the gall bladder wall indicating gangrenous cholecystitis. Patient underwent emergency laparoscopic evaluation and cholecystectomy was done for source reduction.

HISTORY AND SYMPTOMATOLOGY

Some of the chief complaints about GI disorders are pain abdomen, distension of abdomen, vomiting blood, passing blood in the stools, frank bleeding from the rectum, anorexia, vomiting, diarrhea, and constipation. Try to establish the chief complaint, allowing patient to explain one or two symptoms responsible for his/her hospital visit. In case of multiple complaints, narrow down to the most significant complaint that made the patient seek treatment.

Pain abdomen is a common complaint; determine the character of pain, site, severity, radiation of pain, chronology, duration, and associated symptoms. Pain can be characterized as dull aching, sharp, burning, colicky, stabbing, or cramping type. The principal complaint in a peritonitis patient is pain abdomen. The visceral pain arising due to distension or spasm of a hollow organ (e.g., obstruction or cholecystitis), or ischemia, it is dull aching in nature and difficult to localize. However, parietal pain arising from the irritation of the peritoneum (e.g., acute appendicitis) or if an extraperitoneal structures (e.g., muscles) is involved, is sharp, and can be localized easily. In elderly patients and corticosteroids use, acute abdominal conditions have subtle pain with diffuse tenderness. The colicky pain with twisting in nature is observed in organs with physiological peristalsis (e.g., intestinal obstruction, gall stones, and renal stones; **Table 1**).

The location of the pain with respect to the abdominal quadrant can give clue in diagnosis **(Fig. 1)**. The chronology of pain is important in some conditions; acute diverticulitis causes pain in the lower abdomen in the midline, which then shifts to the left lower quadrant (LLQ); whereas in acute appendicitis, pain usually commences in the periumbilical region later relocating to the right lower quadrant (RLQ). It is prudent to access the severity of the objectively by VAS (visual analog scale) or NRS, or behavioral pain assessment scale as a part of standardization of care.

Ascertaining the radiation of pain is important clue for diagnosis. In acute cholecystitis, pain radiates to the right shoulder due to diaphragmatic irritation, whereas pancreatitis pain radiates to the back. Onset of the pain is also important. In case of hollow viscus perforation, pain is acute in nature, usually of maximum severity from the onset. Abdominal aortic aneurysm rupture also presents as sudden shooting abdominal pain (or back pain) associated with hemodynamic collapse or syncope or neurological deficits. Additionally, sudden lower abdominal pain with maximum severity from the onset is observed in ruptured ovarian cyst, ovarian torsion, or testicular torsion. However, gradually increasing pain of insidious nature is observed in hollow viscous obstruction, especially in large bowel obstruction. Mesenteric ischemia causes acute pain, especially initiating in the periumbilical region.

Associated symptoms and aggravating relieving factors are essential part of history-taking in pain abdomen. Pain abdomen, which relieves after an episode of vomiting, usually originates from the upper GI tract. Diarrhea associated with pain abdomen is usually associated with infective/noninfective gastroenteritis/irritable bowel syndrome. Complete intestinal obstruction causes abdominal pain accompanying with constipation or

TABLE 1: Character of pain.

Pain	*Description*
Dull aching (difficult to localize)	Visceral pain due to distension or spasm of a hollow organ (e.g., intestinal obstruction, cholecystitis) and ischemia (e.g., mesenteric ischemia)
Sharp pain (localized easily)	Parietal pain due to irritation of peritoneum (e.g., acute appendicitis) or if an extraperitoneal structures is involved (e.g., muscles)
Colicky pain (twisting in nature)	Organs with physiological peristalsis (e.g., intestinal obstruction, gall stones, renal stones)

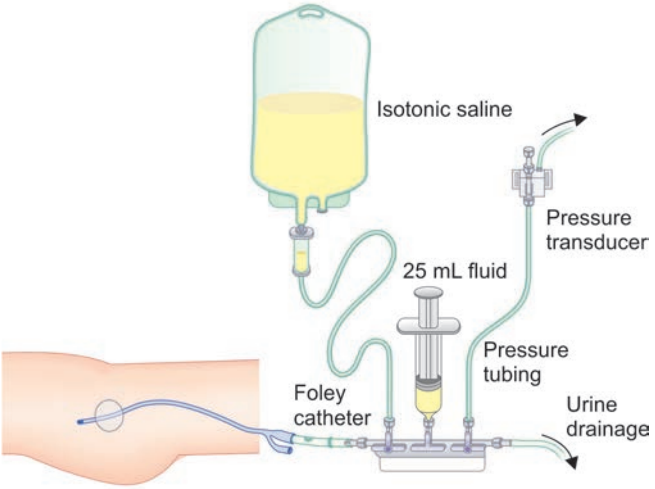


Fig. 1: Measurement of intra-abdominal pressure in intensive care unit (ICU).

obstipation, whereas partial intestinal obstruction can cause paradoxical diarrhea. Besides mesenteric ischemia patients can have diarrhea in the early stages. The use of analgesics such as paracetamol, opioids, nonsteroidal anti-inflammatory drugs (NSAIDs; e.g., aceclofenac) to relieve pain have interfered with evaluation of pain abdomen can cause diagnostic errors.

Abdominal Distension

Distension of the abdomen is conventionally linked with 5 Fs of fat (obesity), fluid (ascites), fetus, flatus (gaseous distension), feces, or "filthy" big tumor. Besides, significance of distension increases if along with the volume there is increase in abdominal pressure. In critically ill, both abdominal volume and pressure increase precipitously due to gut wall edema, ascites, gaseous distention of bowel loops, intestinal dysmotility, and hematoma. In patient with significant abdominal distension, there is manifestation of signs and symptoms such as pain, absence of bowel motions, profuse diarrhea, tenderness, hyperlactatemia, and increased gastric residual volume. This kind of patient deserves further investigations, monitoring, and intervention.

Abdominal distension in a postoperative patient can be an indicator of intra-abdominal bleeding, which deserves timely investigation by serial monitoring of hemoglobin and imaging studies. Furthermore, abdominal distension can also be due to bowel distension as a result of ileus. Besides, in both of these conditions, intra-abdominal pressure (IAP) monitoring and noting down its dynamic changes is essential, as significant increase in IAP can lead to abdominal compartment syndrome (ACS), which not only needs organ support but also requires specific interventions to reduce IAP. Imaging studies can identify bleeding or bowel distension but cannot quantify IAP.

Intra-abdominal hypertension (IAH) is defined as persistent IAP of >12 mm Hg and its classified as per the severity into four grades **(Table 2)**. ACS is a life-threatening condition occurring when a new organ failure is present along with IAH of grade III or IV. Incidence of IAH in ICU is around 50%; however, most of the time it is not acknowledged, whereas ACS occurs in 5–12% of ICU cases.

Intra-abdominal hypertension reduces abdominal perfusion pressure (APP) and when it decreases below 60 mm Hg, it can cause splanchnic hypoperfusion, loss of mucosal integrity, increased permeability, and bacterial translocation. APP is measured by formula APP = MAP – IAP. Renal impairment occurs independent to the reduced cardiac output. Impaired renal perfusion due to the mechanical effect and systemic hypoperfusion leads to increased renin, angiotensin, and aldosterone production, and hence reduced glomerular filtration rate.

TABLE 2: Severity of intra-abdominal pressure (IAP).

Grade	*IAP*
I	12–15 mm Hg
II	16–20 mm Hg
III	21–25 mm Hg
IV	>25 mm Hg
Abdominal compartment syndrome (ACS)	Sustained increase in IAP >20 mm Hg with a new onset of organ dysfunction

To measure IAP, firstly a triple lumen foley catheter is inserted to the patient. To one of its ports, a syringe filled with 25 mL of normal saline is connected. Second port is connected to the pressure transducer through a noncompliant pressure line and zeroed at the midaxillary line. The last port links to the urobag, the proximal part of which is clamped as shown in **Figure 1**. Around 25 mL of normal saline is instilled into the bladder, make sure a part of normal saline instilled flows back up-till the clamp. Finally, IAP is measured with the patient in the supine position, measured at the end of expiration zeroed at mid axillary line. Ensure that the measurement is taken at the end of 30–60 s in the absence of the abdominal contractions.

Gastrointestinal Bleeding

Any bleeding arising from the GI tract lumen, which is visually confirmed by the presence of the blood in the vomitus, gastric aspirate, or stool is termed as GI bleeding (GIB). Upper GI bleed originates from the esophagus till ligament of Treitz at the duodenojejunal flexure and lower GI bleed originates distal to the ligament of Treitz. Asymptomatic bleeding, which is seen only when endoscopy is done for other reasons, is very common in critically ill patients, whereas significant GI mucosal damage, which results in clinically obvious bleeding, is observed in 5–25% of ICU patients. Major bleeding/overt bleeding requiring blood transfusion occurs in 1.5–4% of mechanically ventilated patients. Massive bleeding is termed as when blood loss is >30% of total blood volume, which necessitates 6 or more units of blood transfusion. Severity of GI bleeding is estimated as per **Table 3**.

In history ask for hematemesis (vomiting blood), melena (passage of black tarry stools), hematochezia (passing bright-colored blood in the stools) **(Figs. 2A and B)**, gastroesophageal reflux disease, chronic gastric pain, chronic liver disease, chronic kidney disease, previous history of bleeding, and any previous interventions performed (e.g., endoscopy, surgery for GI bleed, interventional radiological procedures). The presence of comorbidities should be noted especially ischemic heart disease and cardiac failure. Additionally, intake of drugs such as antiplatelets, anticoagulants, NSAIDs, glucocorticoids, and selective serotonin reuptake inhibitors (SSRIs) and alcohol should be noted.

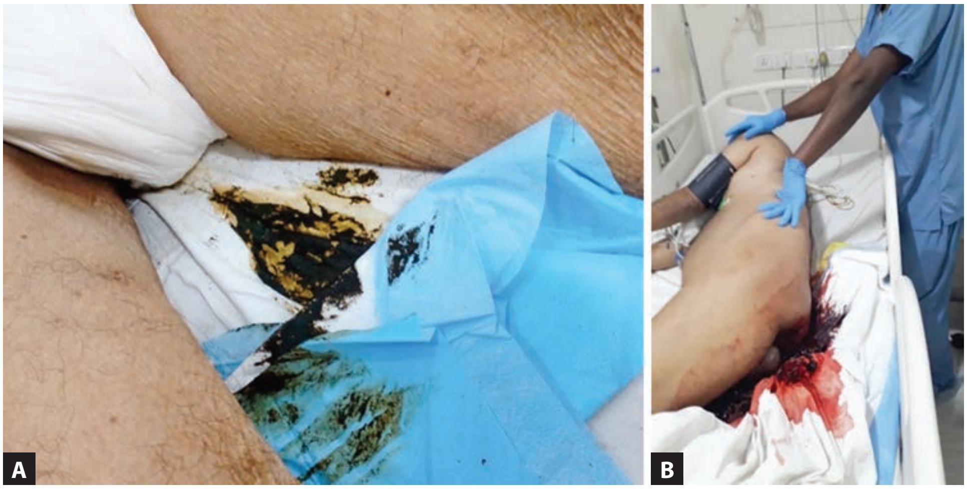
Melena and hematemesis are usually associated with upper GI bleed, whereas hematochezia with lower GI bleed. It is essential to try and establish the source of bleed and the amount of the blood lost. The former is noted by estimating the return from the nasogastric (NG) tube. NG tube insertion causing mucosal damage and in turn leading to further hemorrhage in patients with deranged coagulation is an overvalued concern, as the benefits of insertion with respect to decompressing the stomach, prevention of aspiration, and monitoring; further hemorrhage outweighs its risk. Laboratory tests to be done are complete blood count (CBC), coagulation profile [international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (APTT)], fibrinogen (especially in massive bleeding), renal function test, serum troponin levels, electrocardiogram (ECG), and arterial blood gas (ABG). Hemodynamic assessment to be done by noting pulse rate, blood pressure, and peripheral perfusion (skin temperature, urine output, and consciousness level).

TABLE 3: Severity of upper gastrointestinal bleeding (UGIB) and associated signs.

Physical sign	***Mild***	***Moderate***	***Severe***
Blood loss	<1 L	1–2 L	≥2 L
Blood pressure	Normal	N-borderline	Hypotensive
Orthostasis	No	Possible	Likely
Tachycardia	None-mild	Moderate	Severe
Skin	Warm-perfused	Diaphoretic	Cool, clammy
Urine output	Normal	Diminished	Poor
Sensorium	Alert/anxious	Anxious	Confused/ drowsy

The risk stratification of the patients is done by two scoring systems, first one is Glasgow-Blatchford risk score **(Table 4)**; here a score of >0 is considered high risk, which requires intervention, transfusion, endoscopy, or surgery. The AIMS65 **(Table 5)** is the second scoring system derived from much larger population; it is much simpler to calculate and predicts mortality. Patients who have higher risk of rebleeding and mortality are the ones with hypotension [systolic blood pressure (SBP) <100 mm Hg)], severe comorbidities, INR >1.6, and ongoing bleeding during the time of examination. Causes for the upper and lower GI bleed have been mentioned in **Table 6**.

The primary treatment for significant UGIB due to peptic ulcer disease (PUD) is upper GI endoscopy



Figs. 2A and B: (A) Patient with black tarry stools (melena); (B) Bright-colored blood in the stools hematochezia.

TABLE 4: Glasgow–Blatchford risk score.

BUN	***Score***
<18.2	0
18.2–22.3	+2
22.4–28	+3
28–70	+4
>70	+6
Hemoglobin (g/dL)—for men	
>13	0
12–13	+1
10–12	+3
<10	+6
Hemoglobin (g/dL)—for women	
>12	0
10–12	+1
<10	+6
Systolic blood pressure (mm Hg)	
≥110	0
100–109	+1
90–99	+2
<90	+3
Other criteria	
Pulse rate >100 (per minute)	+1
Malena	+1
Syncope at presentation	+2
Liver disease history	+2
Cardiac failure present	+2

(BUN: blood urea nitrogen)

TABLE 5: AIMS65 scoring system.

Score	***In-hospital mortality***
0	0.3%
1	1.2%
2	5.3%
3	10.3%
4	16.5%
5	24.5%

TABLE 6: Causes for upper and lower gastrointestinal (GI) bleeding.

Upper gastrointestinal bleeding	***Lower gastrointestinal bleeding***
Gastrointestinal origin	Large bowel origin
Esophageal causes: • Esophageal varices • Mallory Weiss tears • Boerhaave syndrome • Esophagitis	• Diverticulitis (17–40%) • Angiodysplasia (9–21%) • Neoplasia (11–14%) • Colitis (infections, inflammatory bowel disease, and ischemic colitis) • Anorectal diseases—hemorrhoids
Gastric causes: • Peptic ulcers (75% of bleeding ulcers) • Gastric varices (10% of varices) • Portal hypertensive gastropathy • Gastritis	Upper GI bleeding presenting as hematochezia (0–11%) *Small bowel origin (2–9%):* • AV malformations • Meckel diverticulum
Duodenal causes: • Duodenal ulcers • Duodenitis	
Other causes: • *AV malformations:* Dieulafoy vascular malformations • *Iatrogenic:* s/p sphincterotomy; s/p duodenal polypectomy • *Trauma:* Facial trauma, sharp object swallowing • Malignancy • Innomino-esophageal fistula • *Swallowed blood:* Epistaxis, hemoptysis, s/p dental surgery	

(AV: arteriovenous)

also known as esophagogastroduodenoscopy (EGD). This is used not only for diagnosis but also to perform endotherapies such as direct adrenaline injections, sclerosing agents, cauterization, clipping, or banding. Additionally, proton pump inhibitors (PPIs) infusion helps in stabilizing clot by keeping pH >6, whereas for adjunctive treatment somatostatin, octreotide, and tranexamic acid can be used. Furthermore, other options that are available for investigation and treatment are computed tomography (CT) angiography, angiographic embolization, or laparotomy.

Variceal bleed originates from the distal esophagus and proximal stomach in the background of chronic liver disease with liver cirrhosis and portal hypertension. UGIB due to variceal bleeding more challenging than PUD.

Some of the endotherapies performed in variceal bleed are rubber band ligation, sclerotherapy, cyanoacrylate glue, or thrombin injections. Massive bleeding, which is refractory in nature, is managed by Balloon tamponade with a Sengstaken-Blakemore, and it is associated with high mortality. Adjunctive therapies, which also help in lowering the risk of rebleed, are terlipressin and octreotide. Lastly, antibiotics usage in variceal bleed reduces mortality.

In UGIB, early endoscopy serves three purposes. Diagnosis is established in 90–95% of cases, performing therapeutic intervention in the variceal and nonvariceal bleed as mentioned in the above paragraph. Finally for risk stratification by establishing the lesion and stigmata accompanying with the lesion. The study of the ulcer base can predict the risk of bleeding by categorizing it according to the Forrest classification **(Table 7)** and Rockall post endoscopy risk score **(Table 8)**. In case of further episodes of bleed, second endoscopy performed within 24 hours can help. Interventional radiological procedures are planned in case of failure of stoppage of bleed with endoscopy. Some of the complications noted with this procedure are bowel ischemia, secondary duodenal stenosis, and gastric, hepatic, and splenic infarcts.

TABLE 7: Forrest classification for upper gastrointestinal bleeding.

Forrest classification		***Rebleeding***
Type 1—Active bleeding		
1a	Spurting hemorrhage	90–100%
1b	Oozing hemorrhage	80–85%
Type 2—Signs of recent bleeding		
2a	Nonbleeding visible vessel	40–50%
2b	Adherent clot on lesion	20–30%
2c	Hematin-covered lesion	5%
Type 3—Lesion without bleeding (flat spot, clean base)		≤3%

Stress-related mucosal damage (SMRD) is present in 60–90% of ICU patients; however, it causes clinical problem in 1.5–8% of cases only. Requirement of mechanical ventilation for >48 hours and coagulopathy are major risk factors for SMRD. Critically ill will develop mucosal damage of the GI system as a result of the gut ischemia, hypotension, vasoconstriction, inflammatory mediators, and high transluminal acidity. Prevention is by stress ulcer prophylaxis with PPIs, H2 receptor blockers, and early initiation of enteral feeding. However, acid suppression has been associated with *Clostridium difficile* infection.

Lower GI bleeding originates mainly from the colon and small intestine distal to the ligament of Treitz. The causes are enumerated in **Table 6**. Hematochezia is the primary complaint; diverticulum is the most common cause followed by angiodysplasia and neoplasia. Massive UGID can present as hematochezia in 12–15% of cases. Colonoscopy is the mainstay of the investigation and management and can identify source of bleed in 74–85% of the cases. Spontaneous remission of massive bleed in observed in up to 80% of the cases.

Nuclear scintigraphy can be utilized whenever available for ongoing bleed, which has diagnostic sensitivity of 86%. It is done by labeling patient's own red blood cells (RBCs) and injecting them into the patient.

TABLE 8: Postendoscopy risk stratification based on the Rockall risk score.

	0	***1***	***2***	***3***
Age (years)	<60	60–79	>79	—
Hemodynamics	• SBP >100 mm Hg • Heart rate <100 beats/min	• SBP >100 • HR >100	SBP <100	—
Comorbidities	No major comorbidities	—	Herat failure, coronary artery disease, any other major comorbidities	• Renal failure • Liver failure • Metastatic malignancy
Endoscopic diagnosis	M-W tear or no lesion identified, and no stigmata of recent hemorrhage	All other diagnosis	Malignancy of upper GI tract	—
Stigmata of recent hemorrhage	No or dark spot only	—	Blood in upper GI tract, adherent clot, visible or spurting vessel	—

(GI: gastrointestinal; SBP: systolic blood pressure)

Technetium-labeled RBC (Tc-RBC) scanning are used, which can detect bleeding rate as low as 0.1–0.5 mL/min. CT angiogram with multidetector CT scans using special protocols and without need for oral contrast can detect ongoing bleed by noticing extravasation of blood intraluminally of the intravenous contrast with diagnostic sensitivity of 90%.

Selective mesenteric angiography using interventional radiological procedures can detect bleed as low as 0.5 mL/min. These advanced intervention procedures can be used both for therapeutic and diagnostic purpose. Using small-sized microcatheters (2.7 mm) and embolizing with microcoils, gel foam, and polyvinyl alcohol, success can be can be achieved in 70–90% and rebleeding rates are as low as 15%. Diverticulum are easily amenable to embolization, whereas angiodysplasia are not. Anatomically, the right colon and cecum pathologies are less amenable to embolization than the left colon. Overall, 10–25% of the cases require surgical intervention.

Diarrhea

There is a wide variation in the definition of diarrhea, most commonly followed one is having three or more loose or liquid stools weighing >200–250 g/day or (>250 mL/day). The World Health Organization defines diarrhea, "as having three or more loose or liquid stools per day, or as having more stools than is normal for that person." Incidence of diarrhea is highly varied in ICU due to lack of proper definition. Reported incidences of diarrhea in ICU varied between 2 and 95%. Bristol stool feces scale classifies stools objectively on the basis of its consistency, with scale 6 and 7 termed as diarrheal stools **(Table 9)**.

History-taking from a patient from diarrhea should include frequency of stools; consistency; color, quantity, and volume of stools; duration of diarrhea; presence of blood or mucus; presence of fat or undigested of foods; drug history; recent food history; travel history; similar history in the family; associated GI symptoms such as pain, nausea, vomiting, tenesmus, jaundice; and systemic symptoms such as fever, chills, and anorexia. It is important to look for signs of dehydration fatigue, thirst, dry skin and lips, dark urine and decreased urine output, headache muscle cramps, dizziness, syncope, orthostatic hypotension, and palpitations. Hypotension is a sign of severe dehydration. Per-rectal examination and examination of stools for blood can give clues to diagnosis. A careful abdominal examination should also be performed to assess for complications (e.g., decreased or absent bowel sounds, abdominal distention, localized or diffuse tenderness, rebound tenderness, and masses).

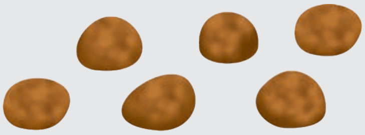
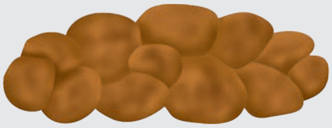
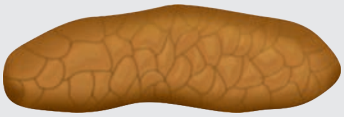
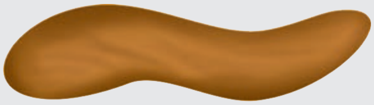
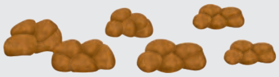
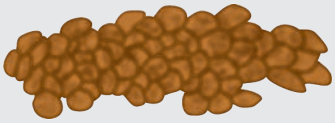
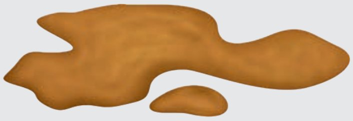
The causes for diarrhea in ICU patients are both infective and noninfective. *Clostridium difficile* is the most common cause for nosocomial diarrhea in ICU with an incidence of 4% and fulminant colitis is observed in 20% of these cases with high mortality of 60%. Diarrhea is associated with electrolyte imbalance, dehydration, perineal excoriation, and in severe cases, it can cause acute kidney injury and hemodynamic instability. Chronic diarrhea is associated with malnutrition.

Vomiting

Vomiting is a forceful retrograde expulsion of gastric contents, whereas regurgitation is the nonforceful expulsion of gastric contents into the esophagus. Nausea is the unpleasant sensation preceding vomiting; nausea is associated with vomiting but not with regurgitation. Sweating, salivation, bradycardia, and reduced respiratory rate due to parasympathetic overactivity are associated with nausea. Vomiting is a nonspecific symptom associated with wide variety of disorders **(Table 10)**. History-taking in vomiting should include duration, its relation to meals, precipitating factors, and contents of the vomitus. Ask for the associated symptoms of pain in abdomen, diarrhea, passage of stools and flatus, headache, and vertigo, history of pregnancy, previous abdominal surgeries, recent intake of any medications, hepatic or renal disease, and cancer (including timing of any chemotherapy or radiation therapy).

Sudden onset of vomiting usually can be of toxic origin, infectious etiology, or associated with central nervous disorder; additionally, acute vomiting (hours to days) can be due to GI inflammatory/structural disorder. Chronic vomiting of intermittent nature can be due to inflammatory disorders. Furthermore, chronic vomiting without systemic symptoms is usually self-induced and chronic nausea without vomiting is usually of psychogenic origin. Vomiting containing recognizable food particles and occurring within hours of intake is due to gastric motility disorders or obstructive disorders at gastric pylorus, whereas small intestinal structural lesion or motility disorders have no relation or inconsistent with timing of food intake.

TABLE 9: The Bristol stool scale.

Type	***Stool picture***	***Consistency***	***Impression***
Type 1		Separate hard lumps—hard to pass stools	Very constipated
Type 2		Sausage shaped but lumpy	Slightly constipated
Type 3		Like a sausage with cracks on surface	Normal
Type 4		Like a sausage or snake, smooth and soft	Normal
Type 5		Soft blobs with clearcut edges easy to pass	Normal—less fiber in diet
Type 6		Fluffy stools with ragged edges, mushy stools	Inflammation
Type 7		Watery, no solid pieces (totally liquid)	Inflammation

In physical examination, look for signs of hypovolemia and abdominal examination of the patient should look for any abdominal scars due to previous surgery; abdominal distension; percuss to note tympany; palpate for abdominal tenderness; peritoneal findings (e.g., guarding, rigidity, and rebound); and any masses, organomegaly, or hernias, auscultate abdomen for bowel sounds (normal, high pitched). In female patients, pelvic examination should be included. Neurologic examination should look for mental status, nystagmus, meningismus (e.g., stiff neck, Kernig sign or Brudzinski sign), and ocular signs of increased intracranial pressure (e.g., papilledema, absence of venous pulsations, 3rd cranial nerve palsy) or subarachnoid hemorrhage (retinal hemorrhage).

Bile-stained vomiting is due to reflux of duodenal contents, which is alkaline in nature and seen due to inflammatory disorders of the stomach. Furthermore, small intestinal obstruction can result in vomiting containing recognizable food particles or clear intestinal secretion based on the level of obstruction. Feculent vomitus can be due to disorders such as gastrocolic or duodenocolic fistula; additionally, it can also occur due to motility disorders leading to stasis and bacterial overgrowth. Projectile vomiting arising immediately after sleep may be due to raise in intracranial pressure, whereas morning nausea and vomiting may be associated with pregnancy. Other causes of the nausea and vomiting acutely occurring may also be associated with intake of medications.

TABLE 10: Different causes for vomiting.

Toxic/metabolic causes	*Gastrointestinal causes*	*Infections*	*Other organ-specific causes*
• Drugs/toxins (alcohol, narcotics, and digitalis) • Biologic toxin (staphylococcal enterotoxin) • Pregnancy • Uremia • Acidosis/ketosis (DKA, alcoholic ketosis) • Electrolyte abnormalities (acute adrenal insufficiency, hypercalcemia)	*Anatomic obstruction or motility disorders:* • Gastric outlet obstruction (peptic ulcer, carcinoma) • Small bowel obstruction (adhesion, herniation) • Colonic obstruction (Carcinoma and volvulus) • Cholecystitis • Idiopathic pseudo-obstruction • Diabetic gastropathy *Inflammatory disorders:* • Gastritis/duodenitis/peptic ulcer disease • Pancreatitis/hepatitis • Peritonitis/mesenteric ischemia	• Viral infections • Sepsis • Meningitis/Encephalitis *Central nervous system:* • Increased ICP (trauma, stroke, meningitis, malignant hypertension, and vascular headache) • Idiopathic epilepsy • Vertigo/motion sickness	*Renal:* • Renal colic • Uremia *Gynecologic:* • Pregnancy • Neoplasm *Cardiopulmonary:* • Inferior wall MI • Congestive cardiac failure • Pulmonary embolism • Post-tussive *Psychogenic:* • Anorexia nervosa/bulimia

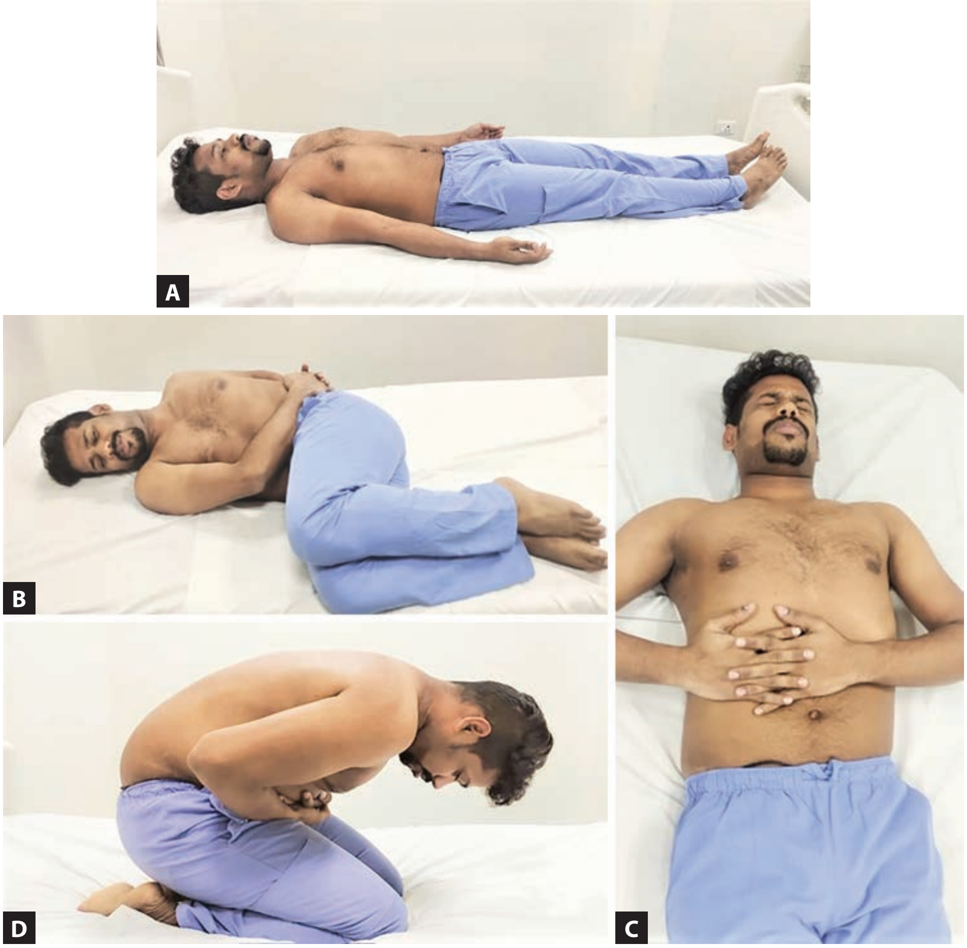
(DKA: diabetic ketoacidosis; ICP: intracranial pressure; MI: myocardial infarction)

OBSERVATION

Physical examination of the abdomen starts with observation of the abdomen; additionally, before starting the examination, preparing the patient and positioning him/her are important. If possible, make sure the bladder is empty before initiating examination. Patient should be in the supine position with extended knees and both the hands placed on the sides or placed on the chest folded, which serves in relaxation of the abdominal muscles **(Fig. 3A)**. Often patient's position can give clue to the diagnosis, patient lying in the fetal position sidewise clinching the abdomen is usually due to a colicky pain (e.g., renal stones; **Fig. 3B**), whereas lying flat with flexed knees and holding on to the abdomen may be clue for spasmodic pain arising from cholelithiasis or obstructive ileus **(Fig. 3C)**; in addition, an anxious, still patient with shallow breathing indicates peritonitis due to intestinal perforation. Furthermore, some patients with pancreatitis or vomiting can be seen in sitting position **(Fig. 3D)**. It is imperative that patient should be assured that assessment will be slow and gentle, besides it always helps in asking the patient to pinpoint with the fingers at the maximum intensity of the pain. Noting the point of maximum pain in a particular quadrant of the abdomen can help in diagnosis as shown in **Figure 4**. The location of an abdominal pain with regard to the abdominal quadrants also helps to narrow the differential diagnosis **(Table 11)**.

Usually, before the inspection of abdomen per se, begin examining the oral cavity, lips, and tongue for ulcers, swelling, symmetry, and leucoplakia. Divide the abdomen into four quadrants with a vertical and horizontal line drawn along the umbilicus as RUQ, left upper quadrant (LUQ), RLQ, and LLQ. Shape and contour of the abdomen should be noted by sitting on the right side of the patient and looking down from the ribcage to pubic bone **(Fig. 5)**, contour can be scaphoid (sunken lower abdomen), flat (athletic), rounded or protuberant (obese), which indicates the nutritional status of the patient and distended abdomen with full flanks is seen in ascites.

Observe for asymmetry of the abdomen by looking across the abdomen side by side or from foot, scanning for bulges or masses. Each quadrant should be carefully examined for any mass and anatomical relationship of the quadrant with the intra-abdominal organ should be considered to establish the cause. The mass with respect to each quadrant can arise due to organomegaly, organ distension, neoplasm, strangulated viscus, or abscess. Upper quadrant mass originates from the stomach or pancreas and the lower quadrant from pelvic organs, distended bladder, or pregnancy. The mass should be examined for its relative motion with respiration, pulsation, and note any peristalsis as it may be due to the distended bowel. In case of bulging flanks, ascites should considered. It is important to differentiate between intra-abdominal mass from a mass originating from abdominal wall. When



Figs. 3A to D: (A) Supine position for abdominal examination; (B) Patient in fetal position sidewise clinching the abdomen due to a colicky pain (e.g., renal stones); (C) Patient lying flat with flexed knees and holding on to the abdomen may be clue for spasmodic pain arising from cholelithiasis or obstructive ileus; (D) Patient with pancreatitis or vomiting can be seen in sitting position.

abdominal muscles are tensed, the mass originating from its wall becomes prominent. Abdominal muscles are made tense by asking the patient to hang his/head off examining table, or hold his/her nose and try to blow or by lifting legs off the table. Abdominal wall masses are due to hernia (umbilical, epigastric, incisional or spigelian), benign or malignant neoplasms, infections, and hematomas.

Generalized distension of the abdomen with or without tenderness can be due to massive ascites (especially if flanks are full), gut perforation with large amount of free air, intra-abdominal hemorrhage due to trauma, ileus, and pancreatitis. However, sometimes even visceral obesity may appear as distended abdomen. A large amount of intra-abdominal fluid, blood, or gas (1–2 L) is required for significant distension to occur. In trauma patients, if distension is substantial then consider massive intra-abdominal hemorrhage.

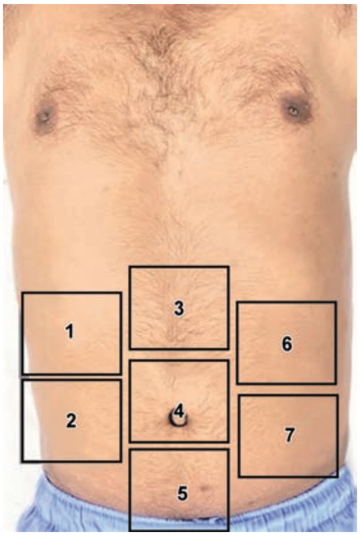


Fig. 4: Abdominal tenderness and point of maximum tenderness.

TABLE 11: Differential diagnosis as per pain in a particular abdominal quadrant.

1. *Right upper quadrant (right hypochondrium):*
    - Acute pancreatitis
    - Acute pyelonephritis
    - Acute viral hepatitis
    - Appendicitis (retrocecal)
    - Basal pneumonia
    - Cholangitis
    - Cholecystitis and hydrops
    - Cholecystolithiasis
    - Duodenal ulcer (perforated)
    - Dyspepsia
    - Liver abscess
    - Liver congestion (in heart failure)
    - Myocardial infarction (inferior)
    - Pericarditis
    - Perihepatitis
    - Pleuritis
    - Renal infarction
    - Spastic colon
    - Herpes zoster (early stage)
2. *Right lower quadrant (lumbar and iliac region):*
    - Adnexitis
    - Appendicitis
    - Bowel obstruction
    - Caecum tumor
    - Diverticulitis
    - Ectopic pregnancy
    - Endometriosis
    - Inflammatory bowel disease
    - Ileitis terminalis (Crohn)
    - Iliac vein thrombosis
    - Infectious enteritis (yersinia, shigella, campylobacter)
    - Inguinal hernia (incarcerated)
    - Irritable bowel syndrome
    - Kidney stones/infarction
    - Meckel diverticulitis
    - Mesenteric adenitis
    - Mittelschmerz (ovulation)
    - Ovarian cyst/torsion
    - Pancreatitis (late stage)
    - Pelvic inflammatory disease
    - Psoas abscess
    - Pyelonephritis
    - Salpingitis
    - Testicle torsion
    - Tubular rupture
    - Tubo-ovarian abscess
    - Ureterolithiasis

Diffuse acute abdomen:
- Acute pancreatitis
- Aortic aneurysm (ruptured)
- Appendicitis (early)
- Bowel obstruction
- Diffuse peritonitis
- Dissecting aorta
- Gastroenteritis
- Irritable bowel syndrome
- Inflammatory bowel disease
- Mesenteric ischemia
- Mesenteric vein occlusion
- Obstructive ileus
- Retroperitoneal hematoma (dorsal)

3. *Epigastrium:*
    - Acute appendicitis (early stage)
    - Acute gastritis
    - Acute pancreatitis
    - Angor abdominal
    - Aortic aneurysm
    - Colon tumor
    - Duodenal ulcer
    - Esophageal spasm
    - Hiatus hernia
    - Myocardial infarction (posterior)
    - Pleuropneumonia
    - Reflux esophagitis
4. *Mesogastrium (periumbilical):*
    - Acute enterocolitis
    - Acute gastroenteritis
    - Aortic aneurysm (ruptured)
    - Appendicitis (early)
    - Bowel obstruction
    - Irritable bowel syndrome
    - Mechanic ileus
    - Mesenteric ischemia
    - Umbilical hernia+
5. *Hypogastrium (suprapubic):*
    - Abortion
    - Acute bladder retention
    - Aortic aneurysm (ruptured)
    - Bladder infection
    - Douglas abscess
    - Endometritis
6. *Left upper quadrant (left hypochondrium):*
    - Acute pancreatitis
    - Acute pyelonephritis
    - Basal pneumonia
    - Colon tumor
    - Duodenal ulcer (perforated)
    - Dyspepsia
    - Gastritis
    - Gastric ulcer
    - Ischemic colitis
    - Jejunal diverticulum
    - Kidney infarction
    - Myocardial infarction
    - Esophageal rupture
    - Pericarditis pleuritis
    - Splenic abscess
    - Splenic infarction
    - Splenic rupture
7. *Left lower quadrant (lumbar and iliac region):*
    - Acute diverticulitis
    - Adnexitis
    - Bladder obstruction
    - Ectopic pregnancy
    - Endometriosis
    - Inguinal hernia
    - Inflammatory bowel disease
    - Irritable bowel syndrome
    - Kidney stones/infarction
    - Mittelschmerz (ovulation)
    - Ovarian cyst/torsion
    - Pelvic inflammatory disease
    - Psoas abscess
    - Salpingitis sigmoid tumor
    - Spontaneous abortion
    - Testicle torsion
    - Tubular rupture
    - Tubo-ovarian abscess
    - Ureterolithiasis

Pseudoacute abdomen:
- Acute intermittent porphyria
- Addison crisis
- Bites (spider, scorpion)
- Diabetic keto-acidosis
- Hemolytic crisis
- Hereditary angioedema
- Henoch-Schönlein purpura
- Herpes zoster
- Hypercalcemia
- Hyperlipidemia
- Lead, iron, ethanol intoxication
- Hyperparathyroidism
- Budd Chiari syndrome
- Costodynia
- Malaria
- Mediterranean fever
- Pancreatitis
- Sickle-cell crisis
- Tabes dorsalis
- Uremic vasculitis

Abdominal movement with respect to respiration should be noted, typically during inspiration, the abdomen moves posteriorly. However, in peritonitis, patient due to localized or generalized rigidity, abdominal movement with inspiration is restricted.

Abdominal skin should be examined for any discolorations, scars, striae, dilated veins, stomas, sinuses, and fistulas. Discolorations may be due to ecchymosis (nontrauma bruising; **Fig. 6A**), bruising (from fresh trauma; **Fig. 6B)**, and erythematous changes. Abdominal ecchymosis (nontraumatic) is usually due to diffusion of blood into the subcutaneous and skin region from underlying peritoneal (hemoperitoneum) or retroperitoneal hemorrhagic conditions. Cullen's sign is periumbilical ecchymosis associated with acute pancreatitis; Grey-Turner sign is bluish discoloration of the flank due to retroperitoneal hemorrhage (hemorrhagic pancreatitis). Similarly, Bryant sign is seen in the scrotum, Fox sign in the proximal inner thigh, and Stabler sign in the inguinal-pubic area.

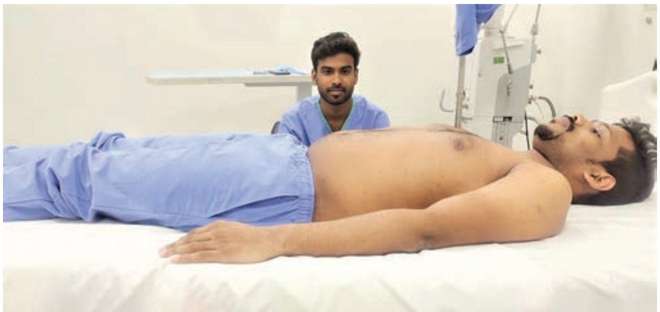
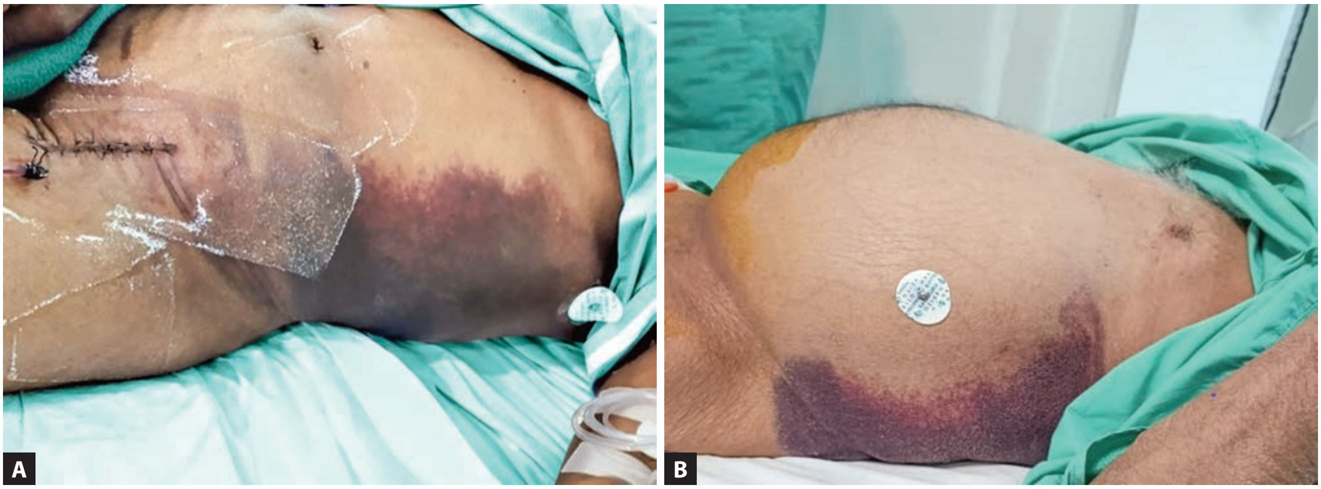


Fig. 5: Shape and contour of the abdomen should be noted by sitting on the right side of patient and looking down from the ribcage to pubic bone.

Abdominal scars usually indicate previous surgeries or trauma and old surgeries are a risk factor for adhesions, obstructions, and infections. The location of the scar can give clue about previous surgeries. Stomas are formed when a part of small or large intestine is brought up to the skin and covered with a stoma bag for collection of stools. Normally, stoma contents are either stool or succus, in case of frank blood or bile **(Fig. 7)**, it is considered as intra-abdominal pathology. Normally stoma is beefy red color, rosette shaped, and connected to the skin at its edges. Look for any discoloration of stoma (dark color—consider ischemia), separation of stoma from the skin, and bulging from its edge (parastomal herniation). Additionally, look for dilated veins on the surface of the abdomen; caput medusa is the dilated vein around the umbilicus observed due to dilated superficial epigastric veins in portal hypertension. Inferior vena cava obstruction makes abdominal veins prominent and there is flow reversal in these veins below the umbilicus.

Auscultation

Auscultation of the abdomen has minor significance as compared in cardiac and pulmonary system evaluation. This particular examination is performed before palpation and percussion as these can interfere with the findings due to disturbance of bowels. Although it is done routinely in ICU, its rarely done properly, hence its clinical



Figs. 6A and B: (A) An intensive care unit (ICU) patient in surgical ICU with ecchymosis developed during in ICU stay with no history trauma; (B) Large ecchymosis on left lateral part of abdomen seen in patient with trivial trauma on long-term steroids.

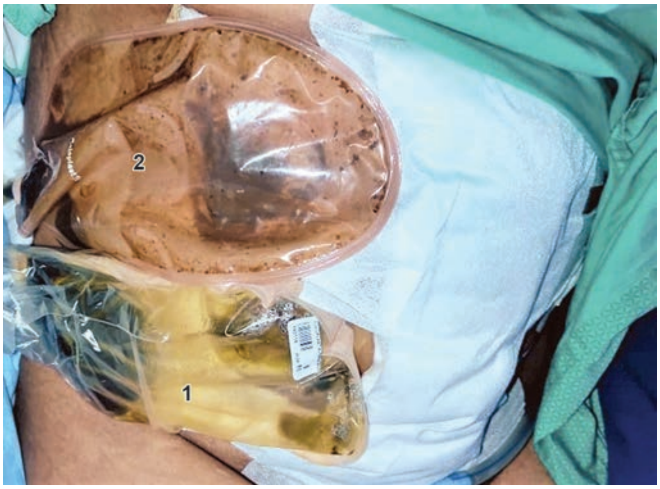


Fig. 7: In a patient with status post exploratory laparotomy/segmental small bowel resection (ileum)/limited right hemicolectomy. (1) End ileostomy—bilious collection and (2) Mucus fistula (associated colon) with mucoserous/sanguineous collection.

importance is often questioned. Place the diaphragm of the stethoscope on the abdomen gently but firmly for auscultation and before examining rub the diaphragm with hands to warm it. Bowel sounds are produced by the peristalsis of the stomach, small intestine, and large intestine bowels due to passage of gas and fluid. These sounds are intermittent, low pitched, and chuckling in nature, heard every 2–5 seconds with some amount of variability. In case of suspected absence of bowel sounds, auscultate for 60 seconds before declaring, however some clinicians advise to auscultate for up to 3 minutes. The abdomen is fairy conductive and bowel sounds are heard equally in all the compartments; however, traditionally the periumbilical region is preferred.

Diminution or absence of bowel sounds are noticed in conditions such as paralytic ileus, intestinal ischemia, or peritonitis. In ICU, studies have mentioned absence of bowel sounds in almost half of the patients, especially ventilated patients on sedatives, opioids, or on inotropic support. Early part of intestinal obstruction or partial intestinal obstruction produces high-pitched hollow tinkling sound similar to the sound of rain on tin roof. Other causes of hyperperistalsis are gastroenteritis and laxatives.

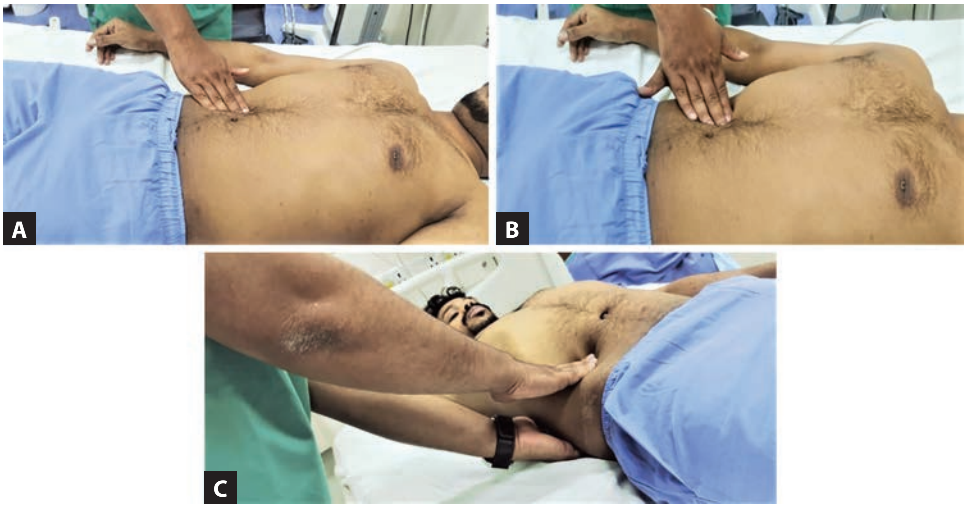
Palpitation

Palpitation is performed after inspection and auscultation, as it keeps patient comfortable and elicits his/her cooperation. It is imperative to notice facial expressions such as grimace, discomfort while palpation. In case of pain abdomen, ask the patient to point at the place with maximum pain. Furthermore, it should be gentle, circular, and always start from the area away from the suspected pathology. It is done initially at superficial levels, here the fingertips are dipped gently into the abdomen, especially to note crepitus due to air or fluid in the subcutaneous tissue **(Fig. 8A)**. It also helps in determining swelling in the abdominal wall such as lipoma or hernias and give a general idea about the tenderness in abdomen.

It is also important to note the abdominal tone right in the beginning stages of palpation; additionally, tenderness is the physical manifestation of pain abdomen. Abdomen is divided into four quadrants **(Fig. 9)** and the additional anatomical sites often mentioned for better clinical detail are epigastric, periumbilical, suprapubic, and McBurney point (located between one-third of the distance from the right anterior superior iliac spine to the umbilicus). Deep palpation is done with two hands **(Fig. 8B)**, the lower hand is firmly placed on the abdomen, whereas the upper hand is used to apply firm but gentle pressure. This method of palpation is used in examining mass per abdomen, eliciting tenderness, and perceiving organomegaly **(Fig. 8C)**.

While eliciting abdominal tenderness, note the quadrant, depth of palpation (superficial or deep), and response from the patient (mild or severe). Abdominal rigidity is an involuntary response due to underlying inflammation (e.g., peritonitis) as a result of spasm of abdominal musculature and it is usually restricted to a particular area of inflammation. However, guarding is voluntary in nature resulting due to generalized abdominal contraction as a way of protecting from pain while palpation. Reassuring the patient and asking him/her to relax the abdomen can reduce guarding. Rebound tenderness is the pain elicited by gently but quickly removing the palpating hand and observe the facial expression of pain and abdominal spasm.

Generalized rigidity and rebound tenderness are two important signs of peritonitis. Whereas, guarding in a trauma patient is a definite sign of intraabdominal solid organ injury or hemorrhage. It has to be noted that abdominal palpation is extremely difficult in patients who are deeply sedated and when patients is given muscle relaxants. In fact, palpation in these patients can give false assurance of normalcy in spite of large hemorrhage especially in abdominal trauma patients.



Figs. 8A to C: (A) Superficial palpation with one hand; (B) Deep palpation using both hands; (C) Bimanual palpation for eliciting kidney tenderness.

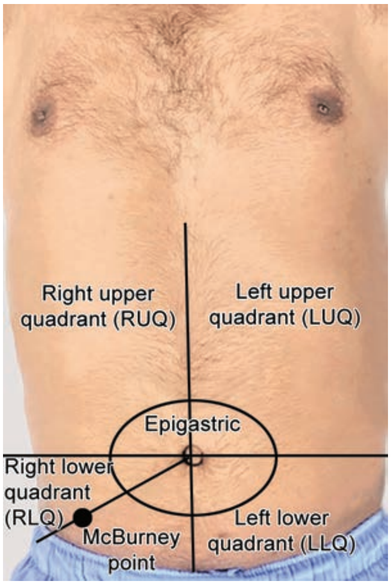

Right upper quadrant (RUQ)
Left upper quadrant (LUQ)
Epigastric
Right lower quadrant (RLQ)
McBurney point
Left lower quadrant (LLQ)

Fig. 9: Four quadrants of the abdomen.

Parietal peritoneum irritation due to inflammation is responsible for tenderness, which when localized to a particular quadrant is due to the pathology of the underlying organ **(Fig. 10)**. RUQ tenderness is noted in cholecystitis, cholangitis, duodenal ulcer, hepatic trauma, hepatitis, stretching of hepatic capsule in hepatomegaly due to right heart failure, or HELLP syndrome. Right quadrant pain, fever, and jaundice are a classical triad of cholangitis. Murphy sign is elicited in cholecystitis when on palpation of RUQ results is a sudden interruption in breath. LUQ tenderness is seen in gastritis, gastric ulcer, splenic infarct, splenic abscess, pancreatitis, sometimes in pyelonephritis, and colon perforation. Epigastric tenderness is elicited in pancreatitis, peptic ulcer disease, lower esophageal pathology, and acute coronary syndrome (ACS). LLQ tenderness is observed in sigmoid diverticulitis, colitis, colonic perforation, incarcerated hernia, ureteric stones, ectopic pregnancy, tubo-ovarian pathology, psoas abscess, and testicular torsion. RLQ tenderness is noted in appendicitis, cecal perforation, incarcerated hernia, ureteric stones, ectopic pregnancy, tubo-ovarian pathology, psoas abscess, and testicular torsion. Generalized tenderness ("board-like" rigidity) is seen in perforated peptic ulcer, including perforated diverticulitis, perforated appendicitis, and necrotic pancreatitis. Finally, flank tenderness is seen in pyelonephritis or perinephric abscess.

Deep palpation is done to note an organomegaly **(Fig. 11)**. RUQ masses are hepatomegaly, gall bladder mass, and carcinoma of the head of the pancreas. LUQ masses palpated are carcinoma stomach and splenic masses. Epigastric masses are pancreatic (pseudocyst or carcinoma), gastric malignancies, and colon malignancies. Lower quadrant masses are sigmoid masses (carcinoma and diverticulosis) on the left and on the right side appendicular abscess, cecal carcinoma, uterine masses, and tubo-ovarian pathologies (on either sides). Pelvic mass in females in fertile age group can be due to gravid uterus. Mass in the flank is palpated by both hands placed on either side of the abdomen due to renal cysts, renal carcinoma, and rarely retroperitoneal masses

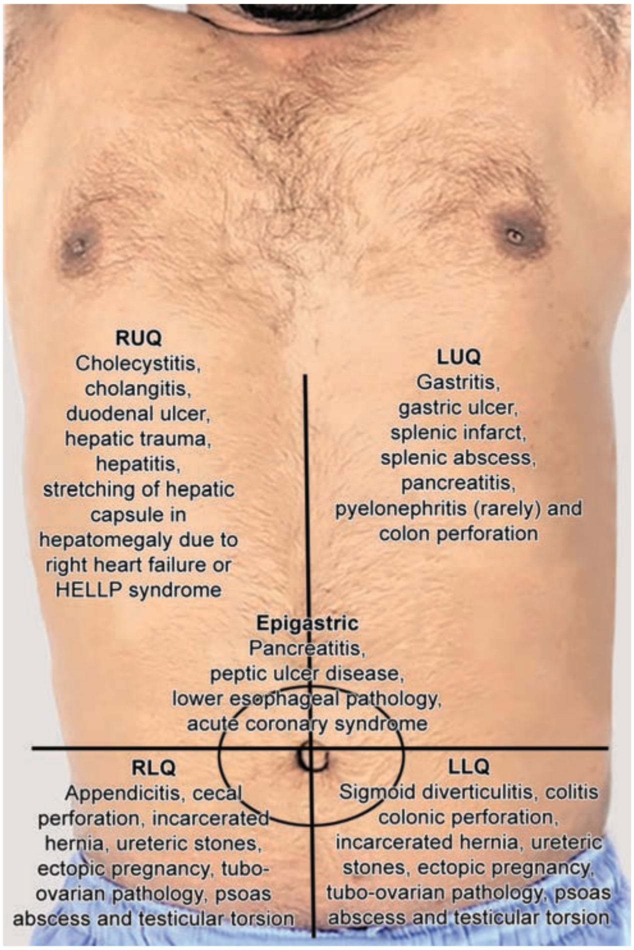

RUQ
Cholecystitis, cholangitis, duodenal ulcer, hepatic trauma, hepatitis, stretching of hepatic capsule in hepatomegaly due to right heart failure or HELLP syndrome
LUQ
Gastritis, gastric ulcer, splenic infarct, splenic abscess, pancreatitis, pyelonephritis (rarely) and colon perforation
Epigastric
Pancreatitis, peptic ulcer disease, lower esophageal pathology, acute coronary syndrome
RLQ
Appendicitis, cecal perforation, incarcerated hernia, ureteric stones, ectopic pregnancy, tubo-ovarian pathology, psoas abscess and testicular torsion
LLQ
Sigmoid diverticulitis, colitis colonic perforation, incarcerated hernia, ureteric stones, ectopic pregnancy, tubo-ovarian pathology, psoas abscess and testicular torsion

Fig. 10: Causes for abdominal tenderness on palpation associated with different compartments of the abdomen. (LLQ: left lower quadrant; LUQ: left upper quadrant; RLQ: right lower quadrant; RUQ: right upper quadrant)

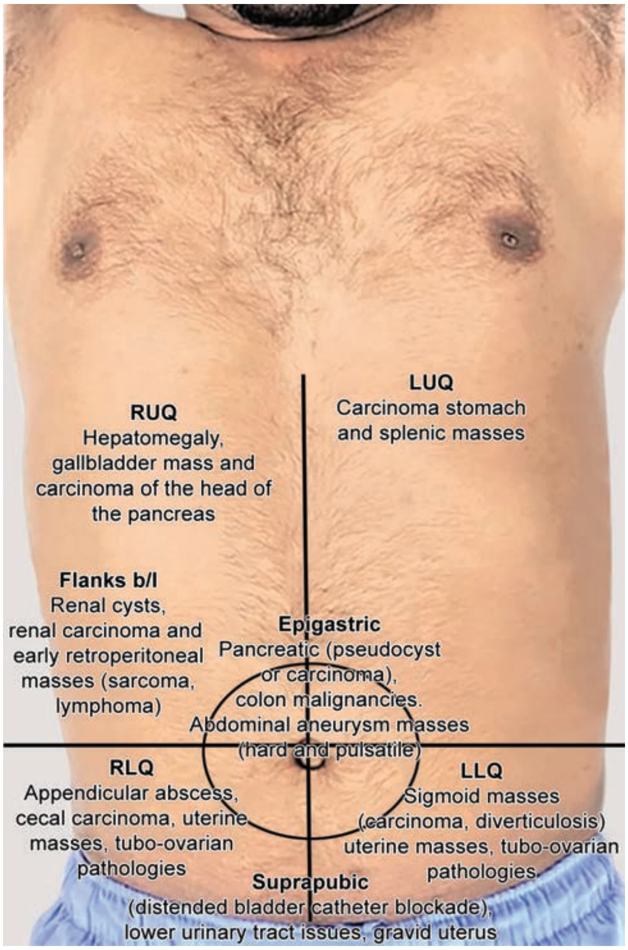

LUQ
Carcinoma stomach and splenic masses
RUQ
Hepatomegaly, gallbladder mass and carcinoma of the head of the pancreas
Flanks b/l
Renal cysts, renal carcinoma and early retroperitoneal masses (sarcoma, lymphoma)
Epigastric
Pancreatic (pseudocyst or carcinoma), colon malignancies. Abdominal aneurysm masses (hard and pulsatile)
RLQ
Appendicular abscess, cecal carcinoma, uterine masses, tubo-ovarian pathologies
LLQ
Sigmoid masses (carcinoma, diverticulosis) uterine masses, tubo-ovarian pathologies
Suprapubic
(distended bladder catheter blockade), lower urinary tract issues, gravid uterus

Fig. 11: Causes for mass per abdomen on palpation associated with different compartments of the abdomen. (LLQ: left lower quadrant; LUQ: left upper quadrant; RLQ: right lower quadrant; RUQ: right upper quadrant)

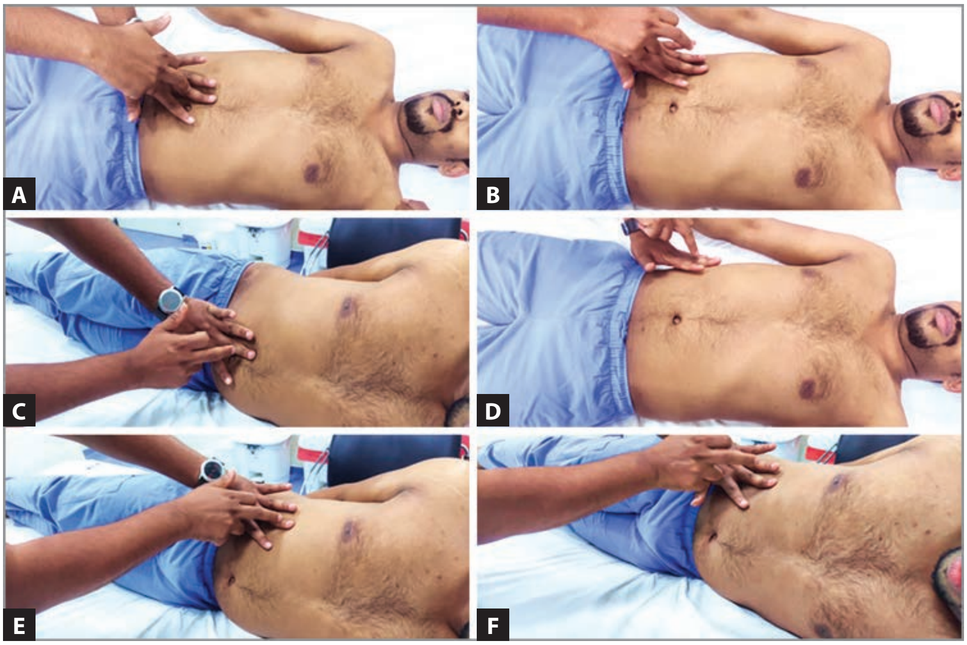
(sarcoma and lymphoma). Suprapubic mass can be due to distended bladder due to catheter blockade and lower urinary tract issues. Abdominal aneurysm masses are hard and pulsatile in nature and located in the epigastric region. Its size is measured by placing index finger on either side of pulsating mass. Abdominal girth measurement has to be done in centimeters/inches with a cleaned tape/disposable tape at the level of umbilicus or at the point of highest distension. It is important to mark the site of measurement and all future measurements have to be made at the same level.

Rectal examination is the part of abdominal examination, in awake and stable patient explain the procedure to the patient, make sure there is reasonable privacy, with due importance given to decorum, respect, and dignity of the patient. Inspect for the rashes, swelling, gluteal asymmetry, perianal abscess, fistulas, areas of fluctuance, and fissures. For rectal examination, place the patient in the left lateral position with knees folded to the chest. Use adequate amount of gel and be gentle with digital examination. Note the tone of the anus, presence of stool, its consistency, and blood stain (in melena or hematochezia), presence of ulcers, polyps, masses, and tenderness should be documented. Hemorrhoids and fissures should be noted. In case of constipation, fecal impaction should be checked and contribution of dyschezia (painful defecation) to this symptom. In male patients, palpate prostate and identify the median sulcus, whereas in female patient, the cervix and lateral fornices should be evaluated. After the examiner's finger is withdrawn, the glove should be inspected for blood and fecal occult blood.

Percussion

Same technique used in chest percussion is used in abdominal percussion. It is done to note intestinal and extraintestinal air and fluid in the peritoneal cavity (ascites). Hyper-resonance (tympany) percussion is noted on distended stomach in the epigastric region and dullness over the liver and enlarged spleen. Hyper-resonance noted on the periumbilical region can be due to gastric distension and due to small intestinal distension, whereas hyperresonance in the LLQ is mostly associated with the ileus due to small intestinal pseudo-obstruction. Colonic pseudo-obstruction leads to hyper-resonance over the entire course of the colon, maximally noted over the RLQ. Hyper-resonance noted in the periumbilical region with dullness in the flanks is very typical of ascites.

Shifting dullness is observed in case of ascites, wherein hyperresonance in the periumbilical region is noted first, then moved slowly percussing toward flanks and observing change of percussion note to dull **(Figs. 12A to F)**. Now roll the patient to the lateral side without lifting the hand



Figs. 12A to F: Pictorial demonstration of various steps in shifting dullness.

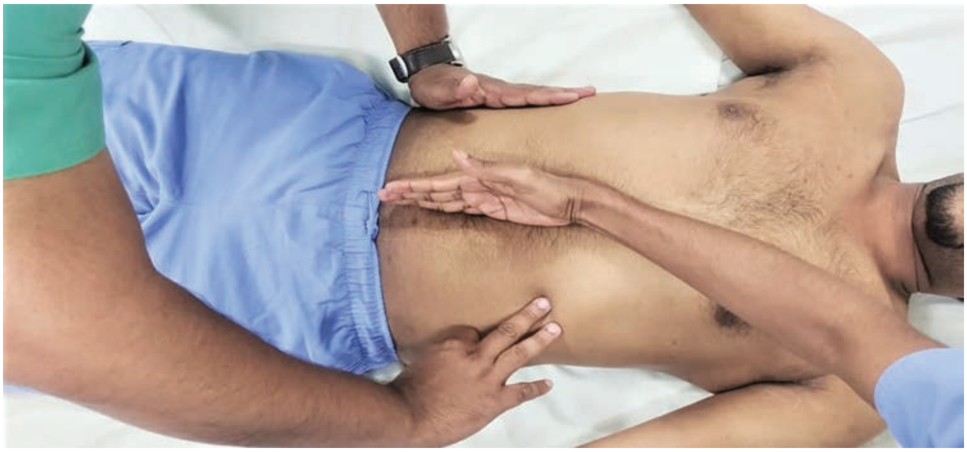


Fig. 13: Pictorial demonstration of fluid thrill (fluid wave test).

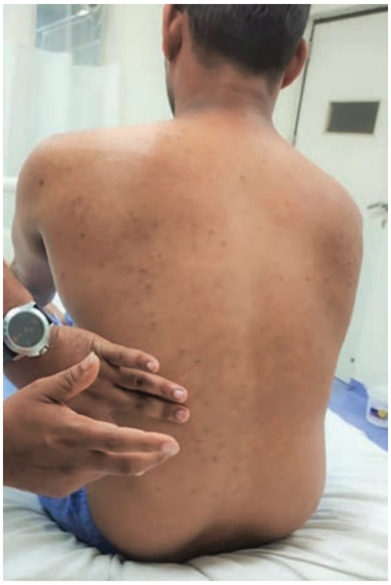


Fig. 14: Percussion on a sitting patient on the right and left costovertebral region can elicit pain in some patients due to pyelonephritis.

and percuss back to the periumbilical region. Now notice the change from hyper-resonance to dullness due to shift in fluid. Fluid thrill test or fluid wave test is elicited by placing the palm on one side of the flank and flicking the finger on the other side, make sure patient (or a bystander) pushes one arm on the midline **(Fig. 13)** to prevent the transfer of the vibrations. In case of ascites, thrill due to movement of the fluid can be felt of the palm of the examiner. Percussion on a sitting patient on the right and left costovertebral region can elicit pain in some patients due to pyelonephritis **(Fig. 14)**.

SUMMARY

Abdomen is considered an enigma. Therefore, profound knowledge of how to conduct a systematic and sequential clinical examination for suspected abdominal conditions is obligatory for day-to-day clinical practice. Integration of laboratory and radiological investigations is equally important in early recognition of abdominal pathologies and for better outcome in patients.

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Chapter

13

Examination of a Critically Ill Immunocompromised Patient

Sunil Karanth, Raghavendra B Goudar

ABSTRACT

In critical care medicine, conducting a thorough history and physical examination is foundational, yet challenging due to the unique environment of the intensive care unit (ICU) and the critical condition of patients. You need structured approach to performing a physical examination in the ICU, considering both planned and focused examinations. Like any case examination of critically ill patients begins with detailed clinical history, emphasis on old medical records, and meticulous clinical examination by paying attention to clinical examination in detail.

INTRODUCTION

Critical care medicine has made progress in leaps and bounds over the last four decades owing to better technology, better understanding of critical illness physiology and its ramifications, and most recently a fundamental shift in the paradigm of treatment with emphasis on "Less is better" and "Do no harm". Despite these advances and new learnings, certain time-tested methods of evaluation cannot be undermined. A thorough history and clinical examination forms the corner stone of this concept even in the critically ill. Eliciting a good history and performing a detailed physical examination in the intensive care unit (ICU) is often a challenge due to the existence of tubes, catheters, lines, and many times abnormal positioning of the patient (such as proning), limitation in attaining an ideal position for examination, distractions from alarms emanating from different medical equipment, other physical distractions in the general environment of the ICU, etc. Besides constraints with time often play a limiting role in ascertaining these two facets of clinical information.

The key to a good physical examination in the ICU would be to interface the patient's clinical condition with the information from the medical technology. The examination of a critically ill patient may often need to be versatile, quick, and efficient. Examination may have two types—planned or focused (often in emergencies or if an unplanned examination is needed).

CASE SCENARIOS

1. *A 45-year-old lady, recently diagnosed to be having carcinoma endometrium, commenced on chemotherapy 1 week ago. Following the chemotherapy, she was discharged and being followed up as an outpatient. She developed abdominal pain and loose stools for one night. She had a brief episode of syncope on the morning of admission, from which she recovered spontaneously and was brought to the emergency department. On evaluation, she was noted to be drowsy, with cold peripheries, tachycardia, and hypotension. Arterial blood gas (ABG) showed increased anion gap metabolic acidosis with elevated lactate. Routine investigations showed hemoglobin 7 g%, a total count of 250/mm^3, and a platelet count of 20,000/mm^3. Chest X-ray was normal, and the echocardiography revealed normal cardiac function. She was diagnosed with neutropenic sepsis with septic shock.*
2. *A 25-year-old gentleman diagnosed to have chronic kidney disease (CKD) secondary to immunoglobulin A (IgA) nephropathy, underwent a live-related renal transplant 1 year ago. Posttransplant he remained stable on three immunosuppressants, namely prednisolone, mycophenolate, and tacrolimus, with which his graft function remained stable. 3 days prior to admission he developed low-grade fever followed by increasing dry cough and dyspnea from the night prior to presentation to the emergency room (ER). On evaluation, he was noted*

to be tachypneic with a respiratory rate of 40 breaths/min saturating 70% on room air. He was commenced on a nonrebreathing mask (NRBM) and subsequently on a high-flow nasal cannula (HFNC) with a flow of 60 L/min and a fraction of inspired oxygen (FiO_2) of 1.0. Screening echocardiography revealed normal ejection fraction (EF) with no evidence of diastolic dysfunction. Lung ultrasound showed B-lines with subpleural shreds. Chest X-ray revealed bilateral "Bat wing" appearance suggestive of pulmonary edema. In view of the history of immunosuppression and the presentation of a short history of hypoxemic respiratory failure, the possibility of pneumocystis pneumonia was suspected.

Thus, the above two cases give a perspective to variations of clinical presentation of immunocompromised patients needing intensive care.

HISTORY TAKING AND SYMPTOMATOLOGY

The basics of history taking largely will remain as in any other specialty of medicine; essentially the symptoms, chronology of events followed by past history, medication history, personal history, etc. However, in many situations, patient presents to the ER may be very ill resulting in constraints on time to elicit a good history. The whole concept of history taking in the ER or ICU when time constraints are present would be focused on complaint-directed history and pattern recognition. It is very important to identify the pattern of a given symptomatology complex and have a structured approach to generate differential diagnoses. Once the initial treatment is done and stabilization performed, it may be appropriate to review the history with the patient or his next of kin including reviewing old reports.

In relation to an immunocompromised patient, it is important to elicit the following additional history:

- *Nature of illness or cause of the immunodeficiency:*
  - *Illness:* For example, diabetes mellitus, retroviral disease, congenital immunodeficiency state or other medical illnesses causing immunodeficiency
  - Ascertain the specific nature of immunodeficiency: For example, cell-mediated or humoral deficiency or both.
- Degree of immunosuppression
- Duration of the immunocompromised state—has a bearing on the type of infective agents causing the illness.

PHYSICAL EXAMINATION

As already mentioned in the previous sections, the physical examination may be planned or focused. First up, let's begin with going about a planned examination followed later by a focused examination in the setting of an emergency or specific problem in the ICU.

- *Planned examination:*
  - This is often the method of examination preferred in daily ICU rounds or on follow-up of patients after the initial resuscitation is completed.
  - Unlike for patients in the wards, it may be advisable to start the ICU ward rounds with the following:
    - Review of the patient's chart
    - Obtain additional history from relevant sources—if appropriate (especially if the history is being revisited or there is a need to obtain more information). The sources of additional information may be:
      - From the patient if in a position to give more history
      - Next of kin
      - Previous hospital referral letter or notes
    - Overview of the patient and his surroundings to get a broad sense of the patient's condition along with its interface with medical technology (including vitals on the monitor, number of infusions, doses of infusions availability of other forms of support like dialysis machine, etc.)
    - Begin by introducing yourself to the patient
  - *Systemic examination:*
    - Begin with the system most appropriate based on the clinical presentation and most likely to have the findings. For example, if the patient presents with a suspected pneumonia, start with respiratory system *or* if the symptoms were localized to the nervous system like seizures, altered sensorium, consider starting with the central nervous system (CNS) examination.
    - Use a structured method for examination to avoid omitting of any findings.
    - *General examination:*
      - Routine evaluation of vitals—heart rate, blood pressure, respiratory rate, etc. Remember to document the need for vasopressors, if the patient needs hemodynamic support.
      - Look for clues which could suggest or indicate specific infections or involvement of organ

  systems or immunodeficiency states. For example:
    - Clubbing
    - Petechial hemorrhage
    - Oral candidiasis
    - Pustules on skin
  - Look for clues to indicate nutritional deficiencies, which could be a common association in many immunocompromised states.
- *CNS examination:* (As a matter of convenience, let's start with CNS—however, in the ICU it would be appropriate to start with the system based on the clinical presentation as described in the previous sections.)
  - Higher mental functions—if practical and considered appropriate
  - *Cranial nerve examination:*
    - Special care to assess the lower cranial nerves (IX, X, XI, and XII), especially if we are considering basal meningitis to be a possible diagnosis
  - Motor system examination
  - Sensory system examination
  - Deep tendon reflexes
  - *Fundus examination:* Very important part of the CNS examination, especially in this subset of patients as it may give important clues to the diagnosis, e.g., tubercles in the choroid and cytomegalovirus (CMV) retinitis
  - Optic nerve sheath diameter (ONSD) using point-of-care ultrasonography—to assess for intracranial pressure (when relevant)
- *Respiratory system examination:*
  - May have limitations in a very sick patient and needs to be supplemented by adequate bedside investigations.
  - Inspection, palpation, percussion, and auscultation
  - Oxygen saturation
  - *Chest X-ray:* Look for patterns suggestive of specific infections
    - For example, cavitatory lesions—suggestive of pulmonary tuberculosis, fungal pneumonia, or necrotizing pneumonia
  - Lung ultrasonography may add vital clues to the nature and extent of involvement.
  - ABG
- *Cardiovascular system examination:*
  - Heart rate and rhythm
  - ECG
  - Inspection, palpation, percussion, and auscultation
  - Bedside transthoracic echocardiography: Hemodynamic parameters, valvular lesions, vegetations, etc.
- *Abdominal examination:*
  - Inspection, palpation, percussion, and auscultation
  - Bedside point-of-care ultrasonography:
    - Free fluid in the peritoneum
    - Any lesions in the solid organs' indicative of abscess or metastatic tumors or primary malignancy of these organs
    - Gallbladder for gallstones and cholecystitis (acalculous cholecystitis may be frequent)
  - Abdominal X-ray—free air or dilated loops of bowel
  - Per-rectal examination: This is a very important aspect of examination, which should not be missed in the immunocompromised host looking for any ischiorectal abscess, prostatitis, etc. as primary source or metastatic source of infections.
- *Genitourinary system:*
  - Renal angle tenderness
  - Examination of external genitalia—lesions to indicate the source of infection or the immunosuppressed state
  - Urine routine and microscopy:
    - For example, pus cells, casts, bacteria, or yeast in the urine
  - Bedside ultrasonographic examination of the kidney, ureter, and urinary bladder:
    - For example, size of the kidneys, pelvicalyceal system dilatation, renal stones, abscesses, and perirenal stranding
    - Bladder wall thickening
- *Hematological system:*
  - Clues to look for any hematopoietic disorder
  - Clinical signs pertaining to this system may be either a consequent of the underlying immunocompromised state or consequent to the infection:
    - Petechiae/ecchymosis/subconjunctival hemorrhage
    - Generalized lymphadenopathy

    - Bedside point-of-care hemogram:
      - Look for anemia, neutropenia, and thrombocytopenia
      - Peripheral smear
- *Unplanned examination:*
  - ICU is a dynamic place. It is very common to be called into the bedside to tackle an acute crisis. As already discussed, a plethora of diagnoses is possible for a given presentation. These situations will need a focused, thoughtful and time-bound evaluation and appropriate response to the crisis.

    These scenarios could include the following:
    - Tachyarrhythmia or bradyarrhythmia
    - Hypotension/hypertension
    - Respiratory distress—new onset of worsening
    - Seizures, loss of consciousness, or new onset of altered sensorium or focal neurologic deficits
    - Sudden onset of chest pain/abdominal pain
    - Changes in urine output, etc.

SUMMARY

In the ICU setting, conducting a physical examination requires adaptability, efficiency, and integration with technological data. While time constraints and patient conditions pose challenges, a systematic approach helps in gathering crucial clinical information for timely management decisions. By balancing comprehensive assessments with targeted evaluations during critical situations, healthcare providers can optimize care delivery for critically ill patients, especially those with immunocompromised states. By meticulously evaluating these aspects during clinical examination, healthcare providers can gather essential information to formulate an accurate diagnosis, assess the severity of illness, and initiate appropriate treatment promptly.

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SECTION 3

Short and Focused Examination

14. **How will I Examine a Patient with Acute Circulatory Failure?**
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15. **How will I Examine a Patient with Hypoxia?**
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16. **How will I Examine a Patient with an Acute Alteration in Sensorium?**
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17. **How will I Examine a Patient with Polytrauma?**
Kapil Gangadhar Zirpe, Balkrishna Dhirajlal Nimavat

18. **How will I Examine a Patient after a Complex Surgery?**
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19. **Burns: Clinical Examination of the Critically Ill Patient**
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20. **How will I Examine a Patient with Neuromuscular Weakness?**
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21. **How will I Examine a Patient with Gastrointestinal Bleeding?**
Justin Aryabhat Gopaldas, Rameshkannan M

22. **How will I Examine a Patient with Acute Gastrointestinal Tract Dysfunction/Acute Abdomen?**
Ajith Kumar AK, Venkatesha Gupta KV

23. **How will I Examine a Patient with Acute Chest Pain in the ICU?**
Sristi Patodia, Raymond Dominic Savio

24. **Examination of a Postcardiac Arrest Survivor**
Anirban Hom Choudhuri, Sakshi Duggal

25. **Examination of a Patient for Difficult Airway in the Intensive Care Unit**
Nishanth Baliga, Sheila Nainan Myatra

26. **Examination of the Critically Ill with Point-of-Care Ultrasound**
Kishore Pichamuthu

27. **Examination of Patients on Extracorporeal Circuit**
Rajesh Mohan Shetty, Rajavardhan Rangappa

28. **How will I Examine a Patient in a Prone Position?**
Sai Saran, Mohan Gurjar

29. **How will I Examine a Patient with Unknown Poisoning?**
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30. **Examination of a Critically Ill Obstetric Patient**
Gauri Saroj, Sandhya Saharan, Dilip R Karnad

Chapter 14

How will I Examine a Patient with Acute Circulatory Failure?

Natesh Prabu R, Vijaya P Patil

ABSTRACT

Acute circulatory failure (shock) characterized by inadequate tissue perfusion is often due to mixed etiologies arising from various disease processes in critically ill patients. Management of a patient with shock requires a multitude of information to understand the causes of shock, active monitoring, and titration of therapies. A detailed history taking and clinical examination will help us understand the patient's circulatory status, etiology of shock, and degree of various organ dysfunctions. It is a challenge to diagnose the exact etiology of shock from a clinical examination of a patient considering the complexity of the illness, organ dysfunction, and sometimes limited access to the patient. This chapter focuses on how to perform a clinical examination of a patient with acute circulatory failure. In addition, the chapter provides an approach to managing patients with acute circulatory failure.

INTRODUCTION

Acute circulatory failure (shock) is a common finding in the intensive care unit (ICU). The patients may present with or develop shock during illness. Most of the patients in general ICU have mixed etiologies of shock, with varied severity (early/compensated vs. late/overt) and may require urgent resuscitation. Recognizing the cause of shock is more important to improve outcomes. A focused history and detailed clinical examination can give much information about the patient's circulatory status, cause of shock, and organ function. The examination should include identifying signs and severity of shock, differentiating shock, and simultaneous assessment of the treatment response. Also, the examination should focus on choosing therapies such as testing for fluid responsiveness and myocardial function to optimize the circulatory status and monitor the response. The clinical assessment should be performed routinely on clinical rounds or whenever necessary to reassess the patients (on demand). In patients with life-threatening shock with very low blood pressure, the assessment and treatment should be done simultaneously to minimize organ damage. In such cases, the treating physician should do a rapid and focused clinical examination since initiating treatment is important. Once the patients improve and reach safe hemodynamic resuscitation targets or in patients with early compensated shock, a detailed examination should be performed. The physician should perform a thorough echocardiographic examination, institute invasive monitoring, understand the hemodynamics status, and plan management.

CASE SCENARIO

A 68-year-old male, weighing 70 kg, presents to the emergency room. He is a known case of uncontrolled diabetes mellitus with an HbA1c of 8.5%, hypertension, and coronary artery disease status. He has undergone percutaneous transluminal coronary angioplasty (PTCA) following an anterior wall myocardial infarction 2 years ago. Additionally, he has dilated cardiomyopathy with an ejection fraction of 45% and New York Heart Association class II symptoms. He also has moderate chronic obstructive pulmonary disease (COPD) with moderate pulmonary arterial hypertension. The patient reports left lower leg swelling for 4 days, accompanied by 3 days of fever, cough, and 5 episodes of loose motions. He also complains of left-sided chest pain and increasing breathlessness over the last 24 hours.

On clinical examination, he appears drowsy but responds to commands. His respiratory rate is 35 breaths/min with the use of accessory muscles, visible jugular venous pulsations, and has bilateral crepitations on auscultation of lung fields. His blood pressure is 82/52 mm Hg; heart rate (HR) is 98 beats/min, and his oxygen saturation is 92% on 10 liters of oxygen per minute. His right leg is cold up to midthigh and has discoloration of the toes. His left leg is red up to the knee, is warm, and blisters are present over his foot. Electrocardiogram shows ST-segment depression and T-wave inversion in anterolateral leads. In the emergency room, his blood cultures are taken, and empirical antibiotics are initiated as per institutional protocol. Due to his deteriorating condition, he was intubated and transferred to the ICU for further management.

In the ICU, the patient was initiated on mechanical ventilation using assist volume control mode with a lung protective strategy [*tidal volume (TV) 6 mL/kg predicted body weight,* FiO_2 *0.6, respiratory rate 30 breaths/min and positive end-expiratory pressure (PEEP) 10* cmH_2O].

On examination, the patient's peripheries were *cold to touch*, and the capillary refill time (CRT) was 5 seconds. *Serum lactate levels were elevated* at 3.2 mmol/L. Blood pressure was 106/60 mm Hg, mean arterial pressure (MAP) was 70 mm Hg on noradrenaline infusion of 0.9 µg/kg/min, and HR was 126 beats/min. Urine output over previous 2 hours was 40 mL/h. The echocardiogram (ECHO) with limited window showed left ventricular hypertrophy, with dilated left atrium and right atrium. He had a moderate left ventricular *systolic dysfunction with ejection fraction of around 30%*, and E/e'–10. Tricuspid annular plane systolic excursion (TAPSE) of 1.6 with moderate tricuspid regurgitation. Lung ultrasonography showed bilateral B lines in axillary and infra-axillary area, positive shreds sign in both posterolateral lung areas (PLAPS point) and minimal pleural effusion. Chest X-ray showed bilateral lower zone haziness. Arterial blood gas analysis revealed a pH of 7.32, PaO_2 of 82 mm Hg, $PaCO_2$ of 37 mm Hg, and bicarbonate (HCO_3^-) level of 19 mmol/L, $ScVO_2$ 64%, Pv-aCO_2 7 mm Hg.

Laboratory investigations revealed the following:
- *Hemoglobin (Hb):* 11.1 g/dL
- *White blood cell (WBC) count:* 18,200 cells/mm^3
- *Platelet count:* 105,000 cells/mm^3
- *Serum creatinine:* 1.89 mg/dL
- Other laboratory parameters were within normal limits.

HISTORY AND SYMPTOMATOLOGY

History in patients with acute circulatory failure is sometimes straightforward and gives diagnostic clues, for example, hypovolemic shock (e.g., multiple episodes of loose motions and/or vomiting), hemorrhagic shock, and severe sepsis, but many times it may not be so obvious. Detailed clinical history plays an important role in such situations. The history should start with the presenting illness, which should be elaborated in detail in chronological order. The history should include the details of events, underlying comorbid conditions, medication history, current details of physiological derangements, organ supports, and assessment and treatment details from the emergency room or previous treating team **(Table 1)**.

GENERAL PHYSICAL EXAMINATION

General examination should be rapid, relevant, and specific. The idea is to quickly recognize the shock states, assess severity, understand the etiology, and identify complications of ongoing therapy. To begin with, look at the vitals and intervene appropriately if the patient is in severe shock before performing a detailed examination. Perform a rapid head-to-toe examination, assessing mental status, pallor, cyanosis, and jaundice followed by an assessment of the adequacy of global perfusion, signs of different shock types and profiles, and their trends followed by a careful look at the bedspace (monitoring devices and ongoing infusions—vasopressors and inotropes) **(Table 2)**.

SYSTEMIC EXAMINATION

The examination of a patient with acute circulatory failure involves the examination of peripheral perfusion, regional microcirculation, detailed examination of cardiovascular system, and other organ systems. The examination should be focused, sequential, and systematic. Many clinical signs are complementary to each other. **Figure 1** provides a pictorial representation of sequential steps to examine a patient with shock and an approach to management.

The examination should help us to:
- Identify shock and differentiate types of shock
- Help to choose therapy and monitor the response to chosen therapy
- Identify possible clues or signs of chronic organ dysfunction that may affect the assessment or choice of therapy and resuscitation targets
- Screen for signs of complications of therapy, for example, fluid overload or severe vasoconstriction due to excess use of vasopressors.

TABLE 1: History and symptomatology—focused on acute circulatory failure.

Domain	***Relevant history to note***	***Comments***
Presenting illness (To identify the presence of shock)	*Altered mental status:* • Altered sensorium • Giddiness, syncope (postural drop in BP in severe hypovolemia) *Low urine output:* • Reduction of urine volume • Any change in color • Concentrated urine—indicate hypovolemia or low cardiac output • Turbid—suggestive of UTI *Associated symptoms:* • Constitutional symptoms—common with sepsis • Cold, clammy extremities • Breathlessness—due to low DO_2 and acidosis because of circulatory failure • Loose motions, vomiting, or volume loss of any cause • Chest pain and heaviness of chest, palpitation (can be due to compensatory tachycardia) • Organ-specific symptoms such as anuria	• It is very important to listen to the patient's symptoms, most of the times the history is given by the caregiver but often patients themselves can explain their symptoms when they have clear mentation. It is important to get the chronology of symptoms that may give clues about the diagnosis. Patients with prerenal causes for oliguria, for example, hypovolemia usually will have a gradual drop in urine output and a change of urine color (darker urine over time) • Symptoms such as lightheadedness and dizziness occur in the early phase of circulatory failure. Drowsiness, agitation, and eventual loss of consciousness are late signs that typically occur when circulatory insufficiency becomes severe and there is cerebral hypoxia **Our patient:** He is drowsy, has cold peripheries, hypotension, discoloration of toes—indicate signs of severe shock
Temporal events (help us to know the likely cause of shock)	• *Current/recent illness:* – *Acute febrile illness:* Fever, myalgia, joint pain – Unilateral pedal edema/bed ridden state with breathlessness—s/o obstructive (PTE) shock – Redness in limbs with blisters, open wounds, or ulcers anywhere on body—s/o septic shock – Recent hospitalization and medications – MI, stroke, vascular insufficiency, etc. • *Recent surgery or instrumentation:* – *Procedures:* Drainage of fluid (pigtail), insertion of tubes/catheters – *Major surgery:* Presence of drain and characteristic of drain fluid postoperative complications such as hemorrhage (pseudoaneurysm/infection) • *Recent trauma:* – Spinal cord injury – Immobility – Fractures • *Deliberate self-harm:* Drug overdose or poisoning	• The history of temporal events is useful to further evaluate for the cause of shock. Hemorrhagic shock is first differential if there is a recent history of surgery or any procedure, patient is on anticoagulants, or is a known case of coagulation disorder • Obstructive shock is likely differential in high-risk patients for venous thromboembolism • Often missed is the history of deliberate intake of drugs or toxins. When there is negative history, suspect myocardial failure due to drug overdose or poisoning **Our patient:** • Poorly controlled diabetic with left leg swelling, redness, blisters were present s/o infection. (Septic etiology for shock) • He also has loose motions (possibility of hypovolemia) • Left chest pain, and discomfort. ECG showed ST changes with past history of coronary revascularization (possible coronary event) • Possible mixed etiology of shock!
Comorbid conditions: • Help us to identify contributory causes, complexity of shock and plan targets of resuscitation	• DM, HTN—autonomic dysfunction, higher BP autoregulatory threshold • CAD, DCM/HF, valvular heart disease: Cardiogenic shock • *Presence of autoimmune disorders/CTDs/peripheral vascular diseases:* May confound assessment or different BP values on both sides	Suboptimal control of comorbid conditions increases the potential risk of organ dysfunction **Our patient:** • Uncontrolled DM (risk of autonomic dysfunction, microangiopathy), HTN (rightward shift of autoregulation curve), CAD, DCM with 40% EF (confound assessment to differentiate septic cardiomyopathy vs. preexisting LV systolic dysfunction)

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Domain	***Relevant history to note***	***Comments***
• Confounders for assessment to choose perfusion targets	• *Malignancies:* Obstructive shock (cardiac tamponade, SVC syndrome are common in these populations) • *Tuberculosis:* Pericardial tamponade and adrenal insufficiency	• Moderate COPD with moderate pulmonary hypertension (challenging assessment—will have elevated right heart pressure, risk of RV dysfunction with fluids, and PEEP). High CVP may influence the perfusion pressure targets (MAP-CVP)
Drugs and medication	• *Anaphylaxis:* New drug, contrast imaging • *Long-time steroids:* Adrenal insufficiency	Anaphylaxis should be suspected when other causes are ruled out and there is temporal association with drugs or radiology contrast administration

(BP: blood pressure; CAD: coronary artery disease; COPD: chronic obstructive pulmonary disease; CVP: central venous pressure; CTD: connective tissue disorder; DCM: dilated cardiomyopathy; DM: diabetes mellitus; DO_2: global oxygen delivery; ECG: electrocardiogram; EF: ejection fraction; HF: heart failure; HTN: hypertension; MAP: mean arterial pressure; MI: myocardial infarction; PEEP: positive end-expiratory pressure; PTE: pulmonary thromboembolism; ST: ST segment in electrocardiogram; SVC: superior vena cava)

TABLE 2: Summary of examination of patients with acute circulatory failure.

Domain	***Eliciting of signs/symptoms (what to look for)***	***Comments***
General appearance	• Pale or grey face à poor/sluggish perfusion s/o very low cardiac output. Commonly seen in profound shock hemorrhagic/obstructive • Flushed face and trunk—s/o vasodilatory state, for example, sepsis **(Fig. 2A)** • Facial congestion (bluish), dilated neck veins: s/o obstructive shock (tension pneumothorax, pericardial tamponade, and SVC obstruction) • *Rash:* Malar rash, viral exanthems, purpura fulminans: sepsis/toxic shock syndrome (refer Chapter 41) • *Sweating:* Commonly seen in palms and forehead (cold clammy skin)	This gives a gross idea of the severity of the circulatory failure and the type of shock
Head and neck	• *Face swelling/periorbital edema:* Subcutaneous emphysema, capillary leak due to inflammation, cellulitis, associated renal/cardiac failure • *Pallor, cyanosis, jaundice:* These give clue about etiology, for example, central cyanosis of cardiorespiratory etiologies, severe hemorrhage, DIC secondary to sepsis • *Oral cavity:* – Examine the undersurface of the tongue, dry mucus membranes suggestive of hypovolemia (tachypnea and mouth breathing can also give rise to dry oral mucosa in absence of hypovolemia) – *Edema of lips:* Anaphylaxis – Oral ulcers (sepsis and CTDs) • Neck pulsations (also upper abdomen)—hyperdynamic circulation • Fundus—retinal signs of CMV/TB in immunocompromised	The examination of the face and neck may give clues about etiology and most of the findings trigger further evaluation. The findings are common and not specific
Chest, abdomen	• Visible cardiac pulsations, scar of previous cardiac surgery—suspect cardiac cause for shock • *Discoloration of abdominal wall:* Intra-abdominal sepsis and retroperitoneal hematoma • Everted umbilicus, venous dilatation over abdomen—liver failure (ACLF) • Skin turgor—dehydration (also seen in extremities) **(Figs. 2A to I)**	This will give an idea of long-standing diseases that may contribute to circulatory failure

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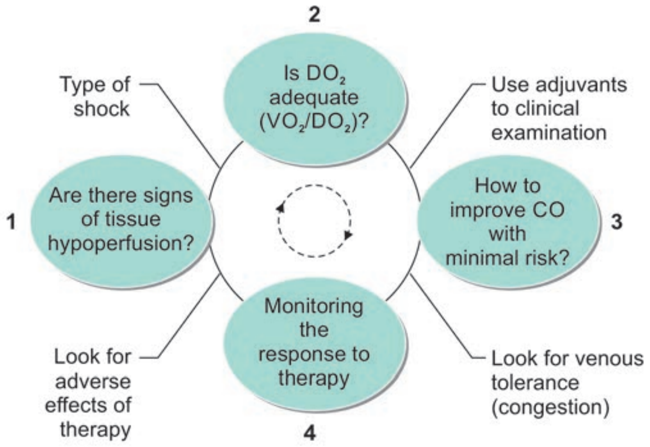
Domain	***Eliciting of signs/symptoms (what to look for)***	***Comments***
Extremities	• Discoloration, peripheral cyanosis/acrocyanosis, and clammy skin • *Fingers and palms:* Nail beds and digits—splinter hemorrhages, digital infarcts—IE, vasculitis • *Peripheral pulse:* All four limbs • Hand and legs: – Pedal edema—pitting edema – *Cellulitis, fasciitis:* Sepsis	• The general examination of extremities is a quick way to identify presence of circulatory failure, complication of vasopressors, and subtle signs of cause of circulatory failure. • Peripheral signs of vasculitis and infective endocarditis are easy to pick up • Cold, clammy extremities—early signs of shock • Mottling, acrocyanosis—late signs of shock
Back and perineum	Excoriations in perineum and pressure sores—frequent loose motions, bed ridden patients	The perineal and back examination is often missed in sick patients that may give clue of source of sepsis
Bedspace and documents	• Vasopressors, inotropes infusions • *Devices:* Cardiac output monitors/ECMO/IABP/temporary pacemakers • *Urine bag:* Urine volume, color	A careful bedspace examination is helpful in getting the trends **(Fig. 2F)**
Systemic examination	To find the source of sepsis, contributory factors to hypotension, severity of organ dysfunction, and assessment of organ function as part of shock evaluation	
Cardiovascular system	• JVP, S3, S4, apex beat position • Tachyarrhythmia or bradyarrhythmias, pulse volume • *Signs of tissue hypoperfusion:* Cold peripheries, discoloration, increased CRT – Vasopressor support • *BP:* Mean BP, diastolic BP, pulse pressure • *Echocardiography:* Pericardial space, chamber size, contractility	Try to judge cardiac output from clinical examination. Assess cardiac function in shock of any etiology. A quick assessment of circulatory system by echocardiography is vital
CNS	• Assessment of sensorium • Can patient protect his airway? • Examination of power, tone, and reflexes • Look for signs of CNS infection	Patients have altered sensorium due to low perfusion to brain and it quickly improves with resuscitation. However, if patient has CNS pathology these signs will persist irrespective of shock resolution
Respiratory system	• Septic focus (e.g., pneumonia, empyema) • Respiratory rate, pattern of breathing, use of accessory muscles (assess work of breathing and need for respiratory support) • Auscultate for adventitious sounds, unequal breath sounds	• Acute circulatory failure can be secondary to severe respiratory infection (source of sepsis) • Pulmonary edema can give rise to rhonchi or crepitations—it is important to differentiate whether these adventitious sounds are secondary to primary lung pathology or cardiac failure, pleural effusion • Circulatory shock is also defined as "a state of whole body oxygen supply demand mismatch"—initiating mechanical ventilation significantly reduce oxygen demand and thus help stabilize failing circulatory system
Gastrointestinal system	• Vomiting and loose motions—volume depletion/abdominal sepsis • *Distended abdomen:* Ascites, ileus, increased intra-abdominal pressure • Tender abdomen	• Ask for frequency and approximate volume of loose stool per day, and enquire for the quality of stool, for example, presence of blood/mucous/watery will help understand pathology • Measuring the loose motions with diaper weight will give a rough idea of amount of stool volume in hospitalized patients. In patients with intra-abdominal pathology, it is important to rule out intra-abdominal hypertension. The common presentation is tense abdomen with oliguria and renal failure

(BP: blood pressure; CNS: central nervous system; CMV: cytomegalovirus; CRT: capillary refill time; CTD: connective tissue disorder; DIC: disseminated intravascular coagulation; ECMO: extracorporeal membrane oxygenation; IABP: intra-arterial balloon pump; JVP: jugular venous pressure; S3: third heart sound; S4: fourth heart sound; SVC: superior vena cava; TB: tuberculosis)


2
Is DO_2 adequate (VO_2/DO_2)?
Type of shock
Use adjuvants to clinical examination
1
Are there signs of tissue hypoperfusion?
How to improve CO with minimal risk?
3
Monitoring the response to therapy
Look for adverse effects of therapy
Look for venous tolerance (congestion)
4

1. CRT, peripheral temperature, skin discoloration, serum lactates, $ScVO_2$
2. Trends of $ScVO_2$, $Pv\text{-}aCO_2$, $Pv\text{-}aCO_2/Ca\text{-}vO_2$, lactate; bedspace, charts
3. Dynamic parameters of fluid responsiveness, ECHO, venous and lung USG, derived parameters from COMs (EVLWI, SVRI, GFI, etc.)
4. Improvement in CO, BP, $ScVO_2$, lactate, and peripheral signs of tissue perfusion

Fig. 1: A pictorial illustration of sequential examination and management of a patient with acute circulatory failure. (BP: blood pressure; CO: cardiac output; COM: cardiac output monitor; CRT: capillary refill time; DO_2: global oxygen delivery; ECHO: echocardiography; EVLWI: extravascular lung water index; GFI: global function index; $Pv\text{-}aCO_2$: venoarterial carbon dioxide gradient; $Pv\text{-}aCO_2/Ca\text{-}vO_2$: venoarterial carbon dioxide gradient over arteriovenous oxygen content; $ScVO_2$: central venous oxygen saturation; SVRI: systemic vascular resistance index; USG: ultrasonography; VO_2: global oxygen consumption)

The first step is to identify the presence of tissue hypoperfusion by clinical examination (supplemented by bedside monitoring parameters), followed by looking for the adequacy of tissue oxygen delivery, improving perfusion (cardiac output) with minimal risk, and monitoring the response to therapy. The clinical examination should be complemented by relevant point-of-care tests, laboratory parameters, echocardiography, and hemodynamic monitoring devices such as cardiac output monitors, microcirculation assessment, and assessment of other organs.

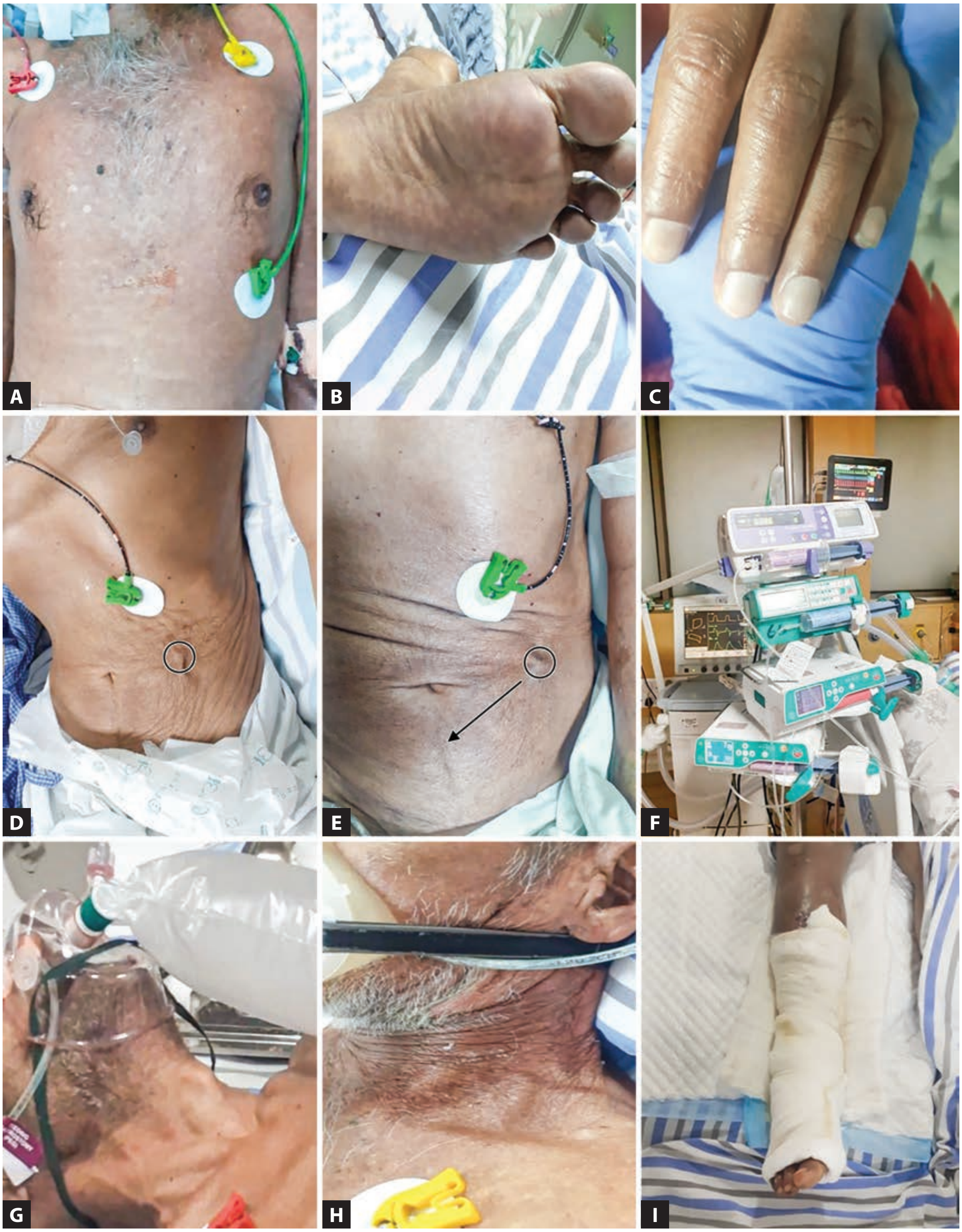
Peripheral Perfusion

Assessment of peripheral perfusion is largely by clinical assessment, which is an easy and quick way to assess at the bedside. It is important to identify patients in the early compensated stage of circulatory failure with careful examination of the patient. In early compensated shock macrohemodynamic parameters such as blood pressure will be normal; however, due to sympathetic vasoconstriction (diversion of blood to central circulation leading to reduced skin perfusion), signs such as cold peripheries, increased core to peripheral temperature gradient, and delayed CRT will be present. Subsequently, as shock advances, compensatory mechanisms begin to fail. As the global blood flow decreases due to reduced cardiac output, the patients will start showing signs of other organ hypoperfusion, often accompanied by a drop in blood pressure.

The commonly assessed signs at the bedside are CRT, mottling score, low urine output, altered mentation, etc. **(Table 3)**. These markers are easy to assess at the bedside but are subjective, and some of these signs may be absent in vasodilatory states such as septic shock or anaphylactic shock (may have warm peripheries). The peripheries and the skin assessment are the starting points to assess peripherical circulation. Other parameters that are measured such as peripheral perfusion index (PPI), central to toe temperature difference (Tc-toe), transcutaneous oxygen/carbon dioxide tension, etc. can be utilized to assess and monitor the peripheral perfusion when available.

Differentiation of Shock

The most important aspect of shock management is to identify the type of shock. Critically ill patients commonly have a mixed etiology of shock. Therefore, it is important to assess all possible causes. History, general examination, and focused assessment supplemented by point-of-care ultrasonography (POCUS), especially echocardiography, usually provide clues to differentiate shock. The history provides the first clue to the possible type of shock. Further examination is targeted to either rule in or rule out the various causes of shock, for example, congestive face, dilated neck veins, and tachycardia are indicators of obstructive shock, while a pale gray face, tachycardia, and thready pulse favor hypovolemic or cardiogenic shock **(Table 4)**. POCUS and echocardiography are handy tools for intensivists that help understand the pathophysiology of shock and also help us understand the response to treatment. For more details, refer to Chapter 26. The sequential approach of focused history and basic clinical examination followed by a close look at invasive arterial pressures, echocardiography, and POCUS provides the necessary information to differentiate shock.



Figs. 2A to I: Few general examination findings in patients with shock. (A) Flushed trunk in a patient with urosepsis; (B) Discolored toes indicating poor perfusion; (C) Pale extremities (it is from a patient with cardiogenic shock with very low cardiac output); (D and E) Demonstrating skin turgor (indicated by the circles) in an elderly patient with hypovolemic shock, (D) poor turgor, and (E) after resuscitation turgor is better, note the distended urinary bladder postresuscitation on E (arrow mark); (F) Patient bed space showing vasopressor infusion and veins; (G) Active respiration, showing active accessory muscles, veins are prominent. This could be due to increased intrathoracic pressure change and may not always indicate increased venous pressure (cardiac pressure); (H) Dilated neck veins in a patient with right ventricular failure. It is good to assess during calm, spontaneous breathing, and in a semirecumbent position; (I) Leg with signs of cellulitis covered by dressing postdebridement, a potential source of sepsis in a patient with septic shock.

TABLE 3: Examination of signs of circulatory failure.

Signs/parameters	***Prerequisites***	***How to examine***	***Comments***
Cold peripheries/central to peripheral temperature difference	• Assess only in covered parts • Peripheries exposed to environment will be cold • Feet are usually covered • Unreliable in patients with preexisting peripheral vascular disease	• Place your hands at the extremities and move toward proximal parts. You will be able to appreciate change in skin temperature. One can also simultaneously assess temperature difference using both hands keeping one hand at extremities (finger/toes) and the other hand toward proximal parts • Examine both sides and compare for discrepancy	• There may be subjectivity and interobserver variability while assessing peripheral coldness • Marking the warm cold junction may help to follow the response to treatment • Use of two temperature probes will be more objective • Coldness of extremities happens due to diversion of blood from skin to central organs (sympathetic vasoconstriction, compensatory mechanism)
Capillary refill time	• Adequate and ambient light • Position of hands at heart level preferably • Use glass slides to appreciate blanching • Chronometer may be more objective to appreciate the refill • Where to assess—finger tips, toes, knee, sternum • Factors affecting CRT— skin temperature, area where it is assessed, amount of pressure given, duration of compression, ambient light. CRT is more in heel compared to finger and knee. Each 1 degree temperature change in skin temperature affect CRT duration	• There are many methods to assess CRT • The sequential steps—compress, release and see response *Technique:* • Apply a pressure at the chosen site for 5–10 seconds, release the compression, and wait for the blood flow to return back to precompression levels • Compress to just achieve blanching and continue same pressure for at least for 5–10 seconds *Pressure applied:* The pressure given can be standardized to ensure uniformity • Apply pressure just to appreciate blanching of nail bed • Apply a firm pressure using glass slide. Increase the pressure till you appreciate blanching of skin and maintain the same pressure **(Figs. 3A to D)** • Use syringe technique as described by Matthias Jacquet-Lagrèze et al. Take 10 mL syringe, pull the piston till 10 mL mark so that it is filled air, cap it or occlude the nozzle with finger. Provide a gentle compression till the piston is displaced to 7 mL mark as shown in picture (compression force = 10 mL to 7 mL). Maintain the force for desired time and release to measure the refill **(Figs. 4A to F)**	During severe shock, the peripheries may have very less blood flow or cyanosed. CRT may not be accurate. In such situations assess CRT in sternum or forehead *Note:* Adequate pressure should be applied for adequate time. Avoid excessive pressure to avoid excessive compression of capillaries

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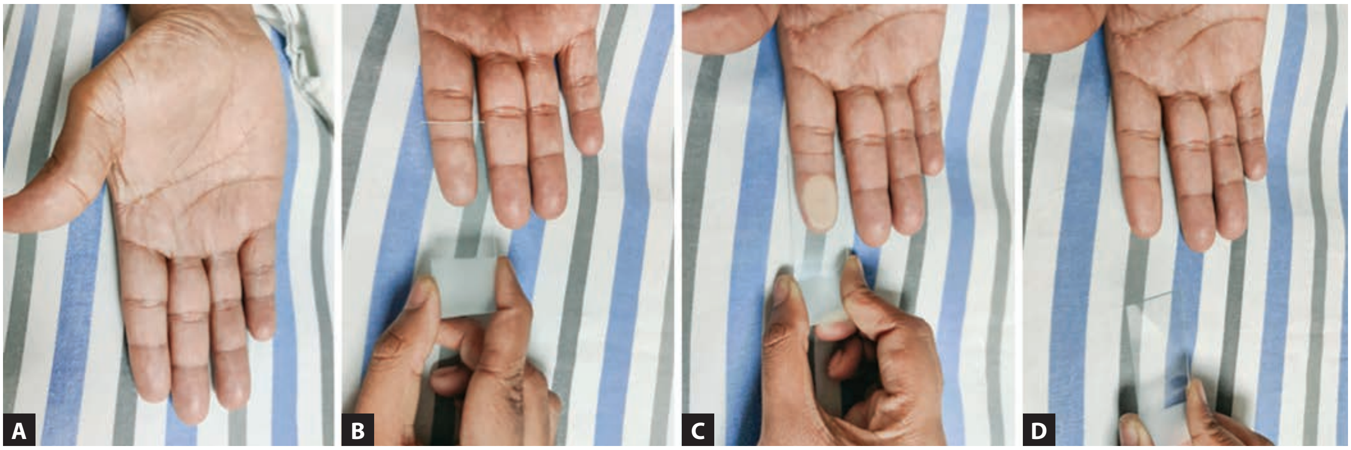
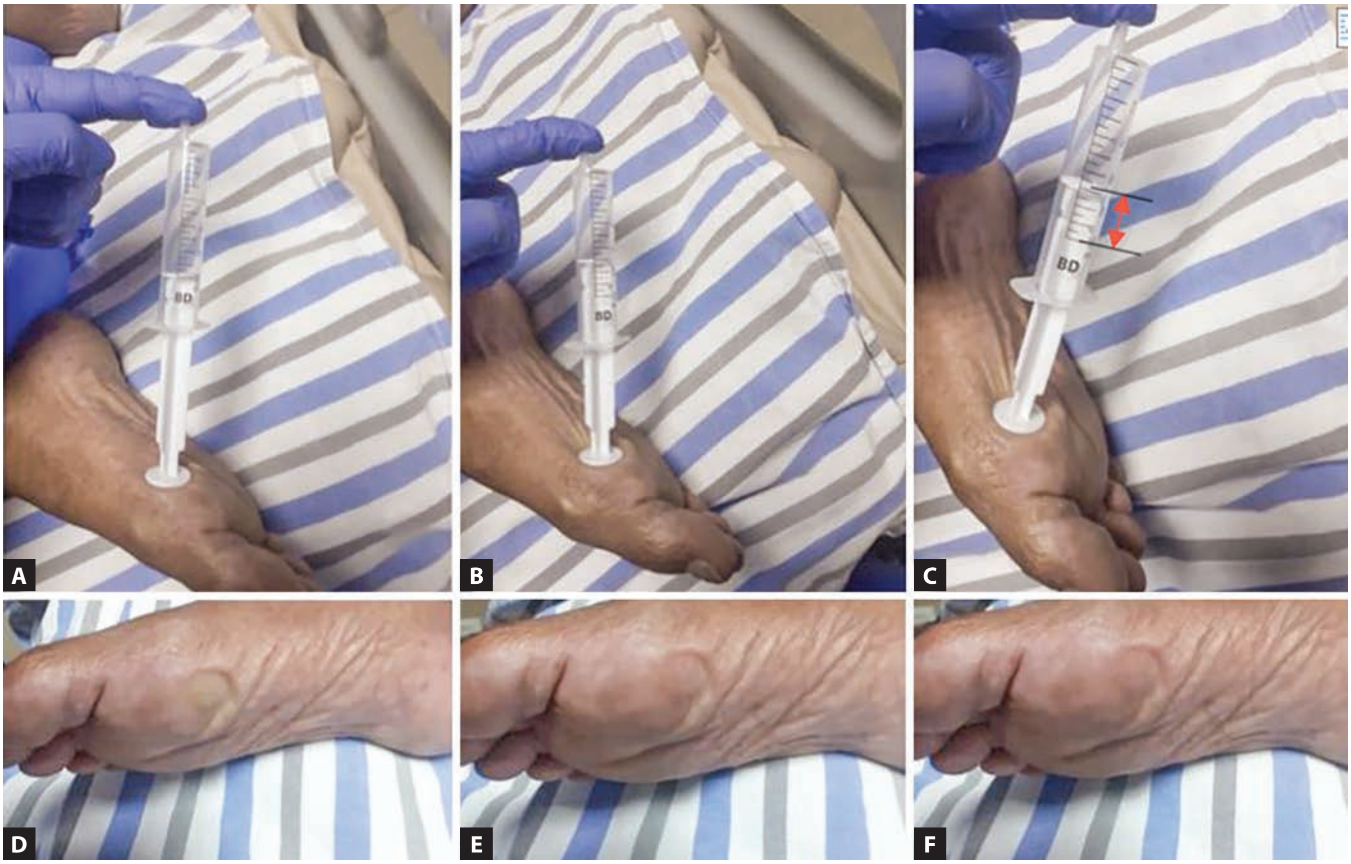
Signs/parameters	***Prerequisites***	***How to examine***	***Comments***
		• How long to give compression—5–10 seconds (average 7 seconds) • How to appreciate the response—using chronometer or by visual estimation (subjective) • Where to measure: Ventral surface of index finger, toes, knee, and sternum	
Mottling of skin	• Adequately exposed limbs • Ambient light	Starts at knee and extends in both cranial and caudal directions **(Figs. 5A to C)**	• More the extent—severe the shock and worse the outcome • May not be appreciated in dark skinned patients
Peripheral cyanosis/ discolorations	Examine all four extremities	Commonly appreciated in toes and sole of foot **(Figs. 5D to F)**	• It is a sign of late stages of shock • Can be due to complication of excessive vasopressor use
Dilated neck veins	• Difficult to appreciate in patients in respiratory distress using accessory muscles • Place patient in a semirecumbent position	Assess during spontaneous breathing	Dilated neck veins: • Just visible (clue to differentiate hypovolemia from obstructive or cardiogenic shock • Dilated—obstructive or cardiogenic shock • Prominent during inspiration and expiration—RVF, pericarditis, restrictive cardiomyopathy • Hepatojugular reflux—reflects heat failure

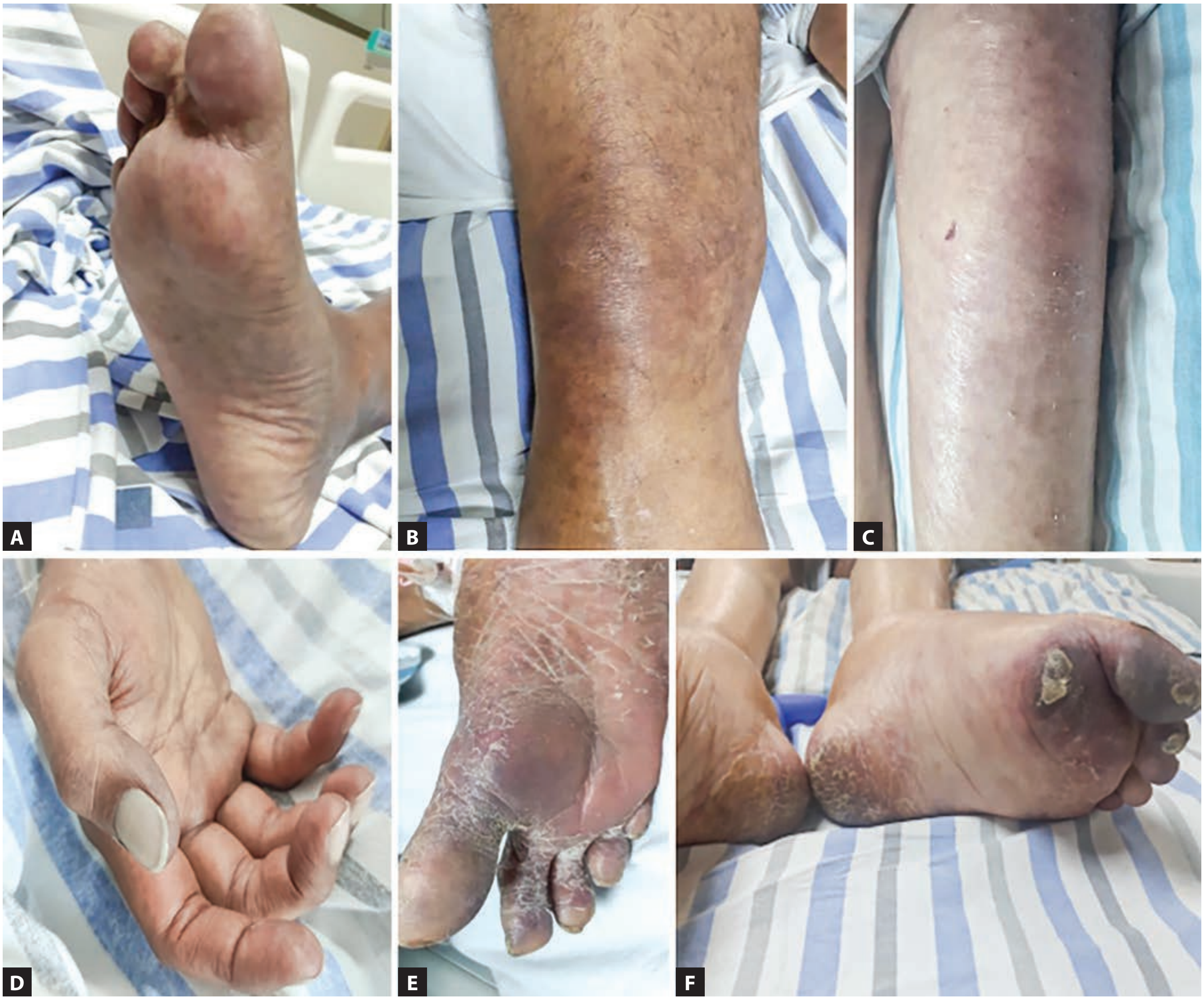
(CRT: capillary refill time; RVF: right ventricular failure)



Figs. 3A to D: Capillary refill test using a glass slide. (A) Position the hand preferably at the level of the heart. Ensure adequate exposure and illumination; (B) Place the glass slide over the ventral surface of the fingertip; (C) Provide a compression force until you appreciate blanching and sustain the compression force for 10 seconds; (D) After releasing the compressive force, note the return of blood flow.



Figs. 4A to F: Capillary refill test using syringe technique. (A) A 10 mL syringe with the piston positioned at the 10 mL mark. The nozzle is occluded with the finger (a cap can be used); (B) Provide compression force till the piston moves to the 7 mL mark; (C) Sustain the compression force for 10 seconds. Note the blanching; (D to F) After the release of compressive force, note the blood flow returning. Note the serial refill from D to F.



Figs. 5A to F: Skin discoloration during different stages of acute circulatory failure. (A to C) Early stages of shock. (A) Skin color change due to poor perfusion; (B) Skin mottling in brown skin patients; (C) Skin mottling in white skin patients. The mottling is better appreciated in white-skinned patient compared to brown or dark-skinned individuals; (D to F) Late stages of shock showing cyanosis of digits.

Adequacy of Tissue Perfusion: Point-of-care Investigations

In most patients, signs of poor tissue perfusion are due to low oxygen delivery in turn due to low cardiac output. The clinical signs of tissue perfusion are commonly assessed to follow up shock dynamics. However, the specific point-of-care laboratory parameter measurements help to understand the adequacy of cardiac output further. Superior vena cava central venous oxygen saturation ($ScVO_2$), arterial-venous partial pressure of carbon dioxide difference (pCO_2 a-v/pCO_2 gap), and serum lactate are commonly measured parameters. Low $ScVO_2$ indicates increased tissue oxygen extraction generally due to inadequate oxygen delivery, which may be due to low oxygen content in arterial blood or low cardiac output, an indicator of tissue hypoxia. A high pCO_2 gap (>6 mm Hg) indicates that improvement in cardiac output will be beneficial. Hyperlactatemia indicates a probable deficit in oxygen delivery at the tissue level and should be closely monitored and interpreted along with other signs. Rather

TABLE 4: Examination to differentiate type of shock.

Clinical	*Echocardiography*	*Adjuvant measurements*
• *Cold and clammy skin:* Classically in hypovolemic (can be present in any low cardiac output state) Peripheries (cold vs. warm—low output vs. high output shock). *See* **Table 2** for details. • Dilated neck veins—(cardiogenic, obstructive vs. hypovolemic or vasodilatory shock) • *Arterial pressures*: – *SAP:* Low—low stroke volume • *Pulse pressure:* – *Low:* Hypovolemic, cardiogenic/obstructive (under-resuscitated septic shock will also have low pulse pressure due to relative hypovolemia) – *High:* Distributive – *DAP:* <40–45 mm Hg (reduced vasomotor tone, e.g., septic shock)	• *Pericardial space:* Tamponade • *Chamber size:* – *Small/kissing papillary muscles:* Hypovolemic – *Normal:* Hypovolemic, obstructive (tension pneumothorax) vasodilatory – *Dilated:* Cardiogenic, septic cardiomyopathy • *Contractility of ventricles:* – Normal or hyperkinetic LV—Hypovolemic/septic/obstructive – Hypokinetic LV: - Cardiogenic (high filling pressures, RWMA) - Septic cardiomyopathy (low filling pressures may be present especially when the patient is not fully resuscitated, commonly global) – *Dilated and hypokinetic RV:* PE/severe ARDS • *Valves:* Stenosis (low cardiac out states), regurgitation • *Valvular vegetations:* Infective endocarditis	• *Cardiac pressure:* – Central venous pressure, PCWP—cardiogenic vs. hypovolemic – E/e′—echocardiographic equivalent of filling pressures • *Myocardial function:* – Echocardiography and cardiac out monitors are vital tools used to assess the myocardial function – Cardiac output monitor: - *SV:* Low hypovolemic/cardiogenic - *SV:* High resuscitated vasodilatory shock - Cardiac function index/global ejection fraction—indices for myocardial performance • *Arterial tone:* – DAP – SVRI
Our patient: He had BP 100/60 mm Hg, moderate left ventricular *systolic dysfunction ejection fraction of around 30%*, and E/e′ 10. Cold peripheries, and just visible JVP/neck veins (i.e., not dilated). Low to normal pulse pressure, with less E/e′ and nondilated neck vein favor possible under resuscitated patient with low cardiac pressures, need further advanced monitoring to further differentiate.		

(DAP: diastolic arterial pressure; E: mitral flow velocity; e′: lateral wall motion tissue Doppler; LV: left ventricle; PCWP: pulmonary capillary wedge pressure; RV: right ventricle; RWMA: regional wall motion abnormality; SAP: systolic arterial pressure; SV: stroke volume; SVRI: systemic vascular resistance index)

than looking at these parameters in isolation, they should be analyzed together.

Measuring the $ScVO_2$ along with the pCO_2 gap helps choose the appropriate therapy. When there is a high $ScVO_2$ (>70%) and high pCO_2 gap (>6 mm Hg), efforts should be made to improve cardiac output if there are other signs suggestive of inadequate perfusion. However, when there is a high $ScVO_2$ (>80%) and low pCO_2 gap (<6 mm Hg), the patient may not benefit from further hemodynamic optimization. Similarly, the pCO_2 gap/$Ca\text{-}vO_2$ ratio, hyperlactatemia, and their trends provide clues about anaerobic metabolism.

Improvement in Cardiac Output with Minimal Risk

While increasing cardiac output is crucial in patients with circulatory failure, over-resuscitation is associated with increased morbidity and mortality. The determinants of cardiac out are preload, contractility, and afterload. It is logical and accepted practice to assess the preload, preload responsiveness, and optimize preload, followed by measures to improve contractility and afterload. The main purpose of fluid infusion is to increase cardiac output, hence the clinical examination should focus on assessing the preload responsiveness in all patients with circulatory failure by choosing the appropriate test of fluid responsiveness. Also, it is equally important to assess the risk of fluid infusion and look for signs of fluid overload. At the bedside, we can assess pressure-based parameters, [central venous pressure/inferior vena cava (IVC) diameter or variability, E/e′ from echocardiography, venous excess ultrasound score], and lung water measurements (lung ultrasonography—B-lines, extravascular lung water index). Clinical signs of fluid overload (edema of extremities) are less reliable **(Table 5)**.

Similarly, monitor for side effects of vasopressors or inotropes—discoloration of nails, digital ischemia, tachyarrhythmias, etc.

TABLE 5: Assessing parameters of fluid overload.

Parameters	***Description***	***Comments***
Clinical parameters		
Pitting edema	• Tissue edema is easy to detect and commonly noticed in patients with fluid overload • Dependent edema is commonly appreciated early over sacrum and later in limbs	Presence of tissue edema is not a specific sign of fluid overload in critically ill patients. It is also commonly seen in patients with increased capillary permeability, for example, systemic inflammation, sepsis
Auscultation of lungs	Crepitation in bases of lungs may be an indicator of pulmonary edema in relevant clinical context	Crepitations can be due to any alveolar pathology such as pneumonia and ARDS
Combined physical signs	Raised JVP, S3, peripheral edema, lung crepitations—combination of these signs indicate fluid overload in absence of cardiac dysfunction	Individual signs have less sensitivity and specificity compared to combination. S3 is more related to heart failure
Cardiac pressure–based parameters		
Cardiac pressures: CVP	High CVP (no definite cutoff but commonly practiced is >15 mm Hg) is always pathological and indicates either fluid overload or poorly compliant heart. Absolute value of CVP is not a marker of fluid responsiveness and is of no value in guiding fluid therapy	• CVP is back pressure for peripheral venous drainage and thus influences organ perfusion pressure (MAP-CVP) • Organ perfusion is influenced by two pressures—forward pressure, which is MAP, and backward pressure, which can be either interstitial pressure or venous pressure. Some organs may have interstitial pressure as back pressure, which may be different than CVP, however in fluid overload, venous pressure acts as back pressure for all organs, and thus CVP can be used as safety variable
Pulmonary capillary occlusion pressure	PAOP is used as surrogate marker of left ventricular function as it measures pressures of left atrium. Like CVP, the PAOP is affected by factors such as compliance of ventricle, preload, variations in intrathoracic pressures, and cannot be used to guide fluid therapy	PA catheter use in ICU is drastically reduced in current era. PAOP measurements help in assessing risk of pulmonary edema, however there is no definite cutoff as the risk also depends on capillary permeability
Ultrasonographic assessments		
Lung USG	• B lines in lung USG—appearance of new B line during resuscitation can indicate pulmonary edema. Studies have shown good correlation with EVLW • Comet tail score (CTS). The sum of all comet tails that are visualized in the ultrasound window. It correlates with check X-ray and EVLWI • The number of B lines on the right and left hemithorax, from second to fourth intercostal space, from parasternal to midaxillary line identify patients with pulmonary edema	Presence of B lines indicates fluid in interstitial space. The fluid (alteration of air-fluid ratio) can be due to hydrostatic (fluid overload, pulmonary edema) or inflammatory process (ARDS, interstitial pneumonia, fibrosis) **Our patient:** Had bilateral B lines and positive shred sign—hydrostatic pulmonary edema vs. ARDS. Needs further evaluation
IVC	IVC variability is not a very good parameter to predict fluid responsiveness irrespective of its size and percentage of variation. Dilated IVC may be a potential indicator of fluid overload along with other parameters	Dilated and nonvariable IVC may not rule out fluid responsiveness or an indicator of fluid overload. IVC diameter >2 cm and <50% variability may be a caution or starting point to evaluate for venous congestion

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Parameters	***Description***	***Comments***
VExUS	• VExUS indicates the venous congestion and severity of congestion • Venous Doppler of hepatic vein, portal vein, and renal vein help to assess the severity of venous congestion	Venous congestion assessment should be done in all patients after initial resuscitation
E/e'	E/A ratio is conventionally used for assessing diastolic dysfunction and E/e' to evaluate left ventricular filling pressure	E/e' <8 generally indicates normal left ventricular filling pressures and >14 indicates elevated filling pressures. The ratio >14 correlates with PCWP >22 mm Hg
Devices-derived parameters and biomarkers		
EVLWI and PVPI	EVLWI represents the amount of fluid accumulated in the interstitial space and affected by increased lung permeability or elevated hydrostatic pressure in the pulmonary capillaries. It is a marker of lung edema and help to guide fluid therapy	EVLWI (measured transpulmonary thermodilution) <7 is normal and >7–10 indicates lung edema. It is used along with pulmonary vascular permeability index (PVPI). PVPI >3.2 indicate increassed permeability as cause for lung edema vs hydrostatic cause. EVLW indicate severity of lung edema, PVPI helps to identify cause for lung edema
BIVA	BIVA estimates body water content. based on the bioelectrical impedance measurements	It is noninvasive. The reliability of measured parameters needs testing
NT-proBNP	NT-proBNP is released from the heart due to increased ventricular wall stress and volume overload. Elevated NT-proBNP levels are associated with left ventricular dysfunction and fluid retention	• NT-proBNP is not a specific marker of fluid overload; it is an indicator of cardiac strain • High value indicates increased wall stress and can indirectly signal hypervolemia when other causes are not present or rule out

(ARDS: acute respiratory distress syndrome; BIVA: bioimpedance vector analysis; CVP: central venous pressure; EVLWI: extravascular lung volume index; ICU: intensive care unit; IVC: inferior vena cava; JVP: jugular venous pressure; MAP: mean arterial pressure; PAOP: pulmonary artery occlusion pressure; PCWP: pulmonary capillary wedge pressure; S3: third heart sound; USG: ultrasonography; VExUS: venous excess ultrasound score)

Monitoring Response to Therapy

It is important to assess the response to the chosen therapy. Clinical examination should focus on examining the parameters that were assessed to initiate the therapy. It is logical to follow-up on the same. Improvement in signs of tissue perfusion, organ function, and a change in vasopressor requirement should be frequently assessed at the bedside. Monitoring the responses to therapy should happen simultaneously during the resuscitation or stabilization phase.

Monitor the Risks and Complications of Therapy

The risk-benefit ratio should be assessed for all therapies we choose. The risk of fluids and vasoactive agents may outweigh the benefits (benefits can be either small or/and transient), so vigilance is essential to identify signs that indicate increased risk or complications during ongoing treatment.

The physician should ask two important questions:

1. Does infusing fluids or vasoactive agents benefit the patient or cause harm? The risk of the chosen therapy should be judged before initiating. Fluid tolerance refers to the degree to which a patient can tolerate the fluid administration without causing organ dysfunction. In contrast, fluid overload refers to an excess amount of fluid accumulated in the body (>10% of body weight). Fluid-intolerant patients commonly develop fluid overload, which may in turn increase the venous pressure leading to venous congestion, which in turn may give rise to organ dysfunction. Though there are no specific clinical signs or parameters that may indicate risk of fluid intolerance, advanced age, underlying multiple comorbidities, and severe

circulatory disturbance on presentation are associated with fluid intolerance.

2. Are there any signs of complications or adverse effects? The assessment should focus on the signs of fluid overload such as pulmonary edema, high venous excess ultrasound score (VExUS) score, and potential complications of vasoactive agents such as peripheral cyanosis, digit ischemia, and tachyarrhythmia.

Clinical Signs of Fluid Overload and Excessive Vasoactive Agents

Clinical signs of fluid overload include tissue edema (limbs, dependent areas—sacrum in ICU patients), anasarca, increased body weight (measurable weighing beds), and organ edema—commonly pulmonary edema. These signs are commonly present when there is fluid overload, but these are not specific, that is, their presence may not indicate fluid overload. Apart from these signs, a few more objective parameters of fluid overload or fluid intolerance are extravascular lung water index (EVLWI), B lines in lung ultrasound (comet-tail images score), VExUS, bioimpedance vector analysis (BIVA), high pulmonary artery occlusion pressure, distended IVC, and raised biomarkers such as brain natriuretic peptide and atrial natriuretic peptide **(Table 5)**.

SUMMARY

Acute circulatory failure is a common concern in seriously ill patients. A comprehensive history and clinical examination play a pivotal role in assessing circulatory status, identifying shock early, and also help understand the underlying cause of shock. During the clinical examination, it is essential to recognize signs of shock, differentiate between various types of shock, and assess the patient's response to treatment. The clinical examination should be supplemented with bedside laboratory parameters, echocardiography, and hemodynamic monitoring devices, such as cardiac output monitors. Additionally, selecting appropriate therapies, such as fluid responsiveness testing and myocardial function assessment, is crucial for optimizing the patient's circulatory status.

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Chapter 15

How will I Examine a Patient with Hypoxia?

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ABSTRACT

Hypoxia is a frequent occurrence in clinically-ill patients and can be life-threatening at times if not recognized and treated promptly. An approach to hypoxia should be systematic with a focus on the patient's clinical condition and use of aids such as pulse oximetry, arterial blood gas analysis, capnography, and point-of-care ultrasonography. The following chapter gives a concise overview of this systematic approach along with a brief management of the common conditions causing hypoxia.

INTRODUCTION

Hypoxemia is defined by a partial pressure of oxygen (PaO_2) of <60 mm Hg. Hypoxemia is not always indicative of tissue hypoxia. Hypoxemia may be the first sign of an impending deterioration, while in some cases, it is easily reversible and correctable.

The symptomatology of a hypoxic patient may be diverse and includes dyspnea, desaturation on pulse oximetry, tachypnea, wheeze, chest pain, palpitations, and respiratory distress. Examination of a hypoxic patient should be quick and systematic as there may be multiple organ systems that may be contributing to hypoxia.

CASE SCENARIO

A 55-year-old male is admitted to your intensive care unit (ICU) with history of swelling of bilateral lower limbs since 10 days, gradually increasing and breathlessness since 2 days. He is a known case of type 2 diabetes mellitus, diabetic nephropathy with a baseline creatinine of 2.6 mg/dL, ischemic heart disease status post percutaneous transluminal coronary angioplasty (PTCA) to left anterior descending (LAD) artery. He is brought to the emergency room where his vitals were as follows: Heart rate—106 bpm; blood pressure—160/96 mm Hg; respiratory rate—28/min; axillary temperature 98.6°F; SpO_2 80% room air. His arterial blood gas (ABG) reveals the following: pH—7.52; PaO_2—52 mm Hg (room air), HCO_3—21.8 mmol/L, $PaCO_2$—28 mm Hg.

How would you go about evaluating this patient?

Detailed evaluation of a patient with hypoxia includes:

- A focused history and physical examination.
- Point-of-care ultrasonography (USG) that should include lung ultrasonography (LUSG), echocardiography, diaphragm USG, and compression ultrasonography (CUS) of the deep veins.
- Assessment of ventilator modes and ventilator graphics.
- Assessment of gas exchange and capnography.
- Additional tests may be needed depending on the suspected etiology.

Each of the aforementioned is discussed in detail in the following text.

HISTORY AND SYMPTOMATOLOGY

The examination of a hypoxic patient should begin with a thorough medical history and physical examination. Many a times, the history may have to be obtained from either the patient's relatives, paramedic, or an accompanying person. It is important to note the reliability of the history obtained.

Prior to history taking and clinical examination, make a quick assessment of the patient's airway, breathing, and circulation status. Ensure the patient is connected to supplemental oxygen to begin with. In case of a threatened airway and evidence of severe hypoxemia, the institution of noninvasive/invasive mechanical ventilation may be needed while assessing for the cause of hypoxemia. Some of the danger clinical signs of severe hypoxemia include visible respiratory distress, usage of accessory muscles of respiration, tachypnea, tachycardia, cyanosis, hemodynamic instability, altered mental status, and low oxygen saturation (SpO$_2$). Scenarios that require emergent intervention for airway management and breathing include upper airway obstruction, tension pneumothorax, etc.

While taking history, do obtain details on patient's age, occupation, status of their activities of daily living (ADL), underlying medical illnesses (comorbidities), personal habits such as smoking or use of recreational drugs. Ask the patient about other symptoms such as chest pain, cough, fever, or recent travel, which may provide additional clues to the underlying cause of hypoxia. **Table 1** gives more information on relevant questions on patient symptoms.

A focused history should be followed by a focused physical exam to identify the cause. **Table 2** highlights the key examination findings in a patient with hypoxia.

TABLE 1: History and symptomatology—focused on acute hypoxia.

S. No.	***Domain***	***Relevant history to note***	***Comments***
1.	Presenting illness—history (To identify the etiology and severity of illness)	• *Cough:* – Onset, duration – Positional variation – Dry/productive cough – *Sputum production:* Color, consistency, frequency, quantity—common in parenchymal lung diseases – *Acute/chronic:* - *Acute cough*—seen with acute conditions, if productive suggestive of infective etiology. Pink frothy sputum is classic of pulmonary edema and rusty sputum is suggestive of pneumococcal pneumonia. Nonproductive cough could be seen with pleural pathologies - *Chronic cough* can occur due to a chronic condition such as COPD, asthma, GERD, or use of medications such as ACE inhibitors – *Hemoptysis:* Streaking of sputum—can occur due to recurrent cough, pneumonia, pulmonary embolism, TB, malignancy - Life-threatening hemoptysis—think of cavitary lesions seen with tubercular or fungal pneumonia, malignancy, pulmonary infarction, diffuse alveolar hemorrhage • *Breathing difficulty (dyspnea):* – Onset, progression, relieving/aggravating factors – History of orthopnea – Paroxysmal nocturnal dyspnea (PND)—suggestive of cardiac etiology—left ventricular failure – Absence of dyspnea in the presence of hypoxemia or "happy hypoxemia" has been described in COVID-19 infection • *Chest pain:* – Onset/duration, site, aggravating/relieving factors – Classical angina may be associated with pulmonary edema – Pleuritic chest pain is typically a sharp pain, increases on cough/deep inspiration – Patients with pneumonia, pneumothorax can also present with chest pain	• It is crucial to pay close attention to the patient's symptoms. When the patient is able to communicate clearly, they can frequently describe their symptoms • But the history is often recorded from the caregiver. It is critical to record the symptoms' chronological order as this could provide diagnostic hints *Our patient:* • Our patient has the comorbidities of diabetes, CKD, and IHD. He is afebrile, admitted with hypoxemia and tachypnea. The following differential diagnoses must be thought of: Acute kidney injury on CKD with fluid overload, left ventricular failure, and atypical pneumonia

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S. No.	***Domain***	***Relevant history to note***	***Comments***
		• *Wheezing:* Wheeze is characteristic of small airway disease and significant wheeze is seen with obstructive airway disease like asthma or COPD • *Altered mental status:* – This is a late sign, indicates severe hypoxemia – The old adage is that a restless patient in the ICU is hypoxic unless proven otherwise • *Associated symptoms:* – Constitutional symptoms such as fever, myalgia, and other prodromal symptoms: common with infective causes of acute respiratory failure – Multiple joint pain, rash, hematuria—evaluate for vasculitis, connective tissue disorders (CTDs) – Abdominal pain, vomiting dysuria—possible intra-abdominal sepsis or urosepsis with possible ARDS. So, organ specific symptoms may help identifying the primary cause and secondary lung involvement – New onset limb weakness, altered speech, difficulty in swallowing—possible neuromuscular causes for hypoxia with type II respiratory failure typically – Sweating, difficult to speak or complete sentences—reflects severity of respiratory compromise – Palpitations—valvular heart disease and heart failure, endocrine causes like thyrotoxicosis with heart failure	• Mild wheeze can be seen in pneumonia, bronchiectasis, and pulmonary edema also • Rule out hypoxia in agitated ICU patients before starting any sedatives for them
2.	Temporal events (Help us to know the likely cause of hypoxia and associated disease)	• *Current/recent illness:* – *Acute febrile illness:* Fever, myalgia, joint pain, and viral prodrome (pneumonia ARDS) – Unilateral pedal edema/bed ridden state with breathlessness—s/o likely pulmonary thromboembolism (PTE) – History related to a specific organ system pathology, e.g., *Skin and soft tissue:* redness in limbs with blisters, open wounds or ulcers anywhere on body; GIT/GUT: vomiting, abdominal pain, dysuria—s/o likely focus of sepsis and associated ARDS – *Recent hospitalization and blood products:* Nosocomial pneumonia/TRALI – Malignancy, chemotherapy, etc. • *Recent surgery or instrumentation:* – *Procedures:* Vascular access in the internal jugular veins or subclavian veins—consider possibility of pneumothorax, hemothorax – Recent procedures such as bronchial artery embolization should raise suspicion of pulmonary infarcts – *Major surgery—thoracic surgeries:* Consider postoperative lung collapse, pneumonia, and bronchopleural fistula – Nonthoracic surgeries like upper abdominal surgeries—basal atelectasis is common due to pain and splinting of diaphragm	• The history of temporal events is useful to further evaluate for the cause of hypoxia. ARDS is the first differential if there is recent history of acute febrile illness, sepsis, and multiorgan dysfunction (MOD) • PTE is likely differential in high-risk patients for venous thromboembolism • Often missed is the history of deliberate intake of drugs or toxins *Our patient:* • Our patient had gradually progressive symptoms of lower limb swelling and shortness of breath (SOB) of short duration. There was no history of fever or prodromal symptoms making an infective cause less likely. Unilateral limb swelling with SOB would have raised the suspicion of venous thromboembolism

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S. No.	***Domain***	***Relevant history to note***	***Comments***
		• *Recent trauma:* – Chest trauma—pulmonary contusions – Rib fractures, flail chest, polytrauma—ARDS – *Immobilization due to major trauma:* DVT – *Long bone fractures:* Respiratory insufficiency may occur as part of fat embolization syndrome • *Recent travel history:* Could be associated with infectious diseases/tropical illnesses such as scrub typhus, dengue, etc. • *History of deliberate self-harm/poisoning:* Drug overdose or poisoning (e.g., paraquat, corrosives, herbicides, etc., that may cause lung damage)	However, bilateral lower limb edema could be attributed to cardiac, renal causes, or hypoalbuminemia due to concurrent chronic illnesses
3.	Comorbid conditions (Help us to identify contributory causes, complexity of hypoxia and plan targets of resuscitation)	• Bronchial asthma, COPD, OSA, OHS—frequent exacerbations • Ischemic heart disease, dilated cardiomyopathy, heart failure, and valvular heart disease—likely risk for pulmonary edema • Presence of autoimmune disorders/CTDs such as rheumatoid arthritis, SLE—possibility of ILD • Malignancies—risk of PTE, chemotherapy related pulmonary toxicity, and pulmonary metastasis • ILDs, chronic pulmonary diseases—patients may have low SpO_2 as a baseline value • Retroviral diseases immunocompromised states—opportunistic infections of lung such as *Pneumocystis jirovecii* pneumonia	• If the patients have chronic respiratory diseases, consider exacerbations of their underlying diseases (lung, kidney, and heart). Suboptimal control of comorbid conditions increases the potential risk of exacerbations, organ dysfunction • The information of comorbid conditions and their control may be helpful to choose target SpO_2, oxygen and BiPAP settings in patients on home oxygen/BiPAP support *Our patient:* • In the case above, the patient is a known diabetic, has chronic kidney disease, and a past history of ischemic heart disease. It is important to take history of baseline activities of daily living, medication history, and compliance to medications • Consider possible diagnosis of heart failure, pulmonary edema, and fluid overload with above history and presentation
4.	Drugs and medication history	• *Anaphylaxis:* Drugs, contrast exposure—bronchospasm, airway edema • Drug related—direct toxicities caused by chemotherapeutic drugs (bleomycin, mitomycin, and cytarabine), amiodarone • Methemoglobinemia due to sulfa drugs, antimalarials, local anesthetics like prilocaine, benzocaine as may confounds SpO_2 interpretation	Anaphylaxis should be suspected when other causes are ruled out and there is temporal association with drugs or radiology contrast administration
5.	Personal/social/ occupational	• Smoking—consider COPD, acute bronchitis, and malignancy • Occupational exposure to dust (asbestosis, silicosis)—pulmonary fibrosis Symptoms would be waxing and waning with shifts/weekends • Tuberculosis in healthcare settings	

(ACE: angiotensin-converting enzyme; ARDS: acute respiratory distress syndrome; BiPAP: biphasic positive airway pressure; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; DVT: deep vein thrombosis; GERD: gastroesophageal reflux disease; GIT: gastrointestinal tract; GUT: genitourinary tract; ICU: intensive care unit; IHD: ischemic heart disease; ILD: interstitial lung disease; OHS: obesity hypoventilation syndrome; OSA: obstructive sleep apnea; SLE: systemic lupus erythematosus; TB: tuberculosis; TRALI: transfusion-related acute lung injury)

TABLE 2: Signs and symptoms to elicit in a patient presenting with hypoxia.

S. No.	*Domain*	*Eliciting of signs/symptoms (what to look for)*	*Comments*
1.	General appearance	• *Facies:* Pursed lip breathing in COPD, may have a flushed look (pink puffers) in emphysema, blue bloaters (overweight, cyanotic patients) seen with chronic bronchitis • Tripod position, sitting upright—consider obstructive and restrictive lung diseases, respectively • *Chest shape:* Kyphoscoliosis, pigeon chest—reduced lung capacities. Barrel shaped chest—COPD, emphysema **(Table 3)** • Flushed face and trunk—s/o sepsis and pulmonary source or ARDS • Body habitus—obese (OSA), thin (COPD), recent loss of weight could indicate chronic illness, malignancy • *Rash—malar rash in SLE, viral exanthems, purpura fulminans:* Sepsis/toxic shock syndrome • *Sweating:* Commonly seen in palms and forehead (cold clammy skin) • *Breathing pattern:* Paradoxical—airway obstruction, spinal cord injury	• This gives a gross idea of the severity of cause of hypoxia and pulmonary compromise • Patients in respiratory distress are often orthopneic and prefer to be in a head up position
2.	Head and neck	• *Facial swelling/periorbital edema/congestion:* Subcutaneous emphysema and tension pneumothorax, SVC obstruction, pericardial tamponade, and associated renal/cardiac failure • *Cyanosis:* These give clue about etiology, e.g., central cyanosis of cardiorespiratory etiologies. Examine the lips and undersurface of the tongue for central cyanosis • *Oral cavity:* – Edema of lips—angioedema, anaphylaxis – Oral candidiasis—HIV/Immunocompromised states • Lymph nodes—tuberculosis (TB), malignancy • Neck pulsations (also upper abdomen)—hyperdynamic circulation • *Fundoscopy:* Signs of cytomegalovirus (CMV), TB in immunocompromised	The examination of the face and neck may give clues about etiology and most of the findings trigger further evaluation. The findings are common and not specific
3.	Chest, abdomen	• *Asymmetrical chest:* Kyphoscoliosis (restrictive lung diseases), abnormal or decreased chest movements—pleural/parenchymal disorders, unilateral expansion—endobronchial intubation, pneumothorax/pleural effusions • Abrasions, contusions—trauma (rib fractures) • Visible scar of previous cardiac thoracic surgery—congenital or valvular heart diseases—pulmonary edema • *Thoracic surgery:* Chronic lung diseases (decortication for empyema, lobectomy, or pneumonectomy for bronchiectasis, malignancy) • *Distended abdomen:* Intra-abdominal hypertension, splinting of diaphragm due to abdominal pathologies	This will give an idea of past respiratory illness and treatment received. Also, give a clue of possible reduced respiratory reserve and potential contributors for current illness and hypoxia
4.	Extremities	• Look for clubbing, peripheral cyanosis/acrocyanosis • Capillary refill time >3 seconds indicates hypoperfusion as a cause of hypoxemia • *Fingers and palms:* – *Nail beds and digits:* Splinter hemorrhages, digital infarcts—infective endocarditis, vasculitis • *Hand and legs:* – Pedal edema—pitting edema – Skin/soft tissue infections like cellulitis, necrotizing fasciitis—source of sepsis	The general examination of extremities is a quick way to identify preexisting cardiorespiratory compromise

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S. No.	*Domain*	*Eliciting of signs/symptoms (what to look for)*	*Comments*
5.	Back and perineum	• Examine back for any sacral pitting edema • Presence of infected pressure sores, perianal ulcers • *Per rectal examination:* Lax sphincters—seen in spinal cord pathologies	The perineal and back examination is often missed May provide subtle clue for possible source of sepsis (infected pressure sore, perineal abscess, etc.)
6.	Bedspace and documents	• *Ventilator screen:* Graphics and derived parameters • Ongoing vasopressors/inotropes infusions • *Devices:* Esophageal pressure monitoring, electrical impedance tomography • ICD bag—volume, content **(Figs. 1A and B)**	A careful bedspace examination is helpful in getting the trends
Systemic examination		To find the source of sepsis, contributory factors to hypotension, severity of organ dysfunction and assessment of organ function as part of shock evaluation	
7.	Cardiovascular system	• Look for elevated jugular venous pulsations, presence of third and fourth heart sounds (S3/S4), murmurs, shift in cardiac apical position • Tachyarrhythmia or bradyarrhythmias, pulse volume—rheumatic heart disease/HF may decompensate leading to pulmonary edema • *Signs of tissue hypoperfusion:* Cold peripheries, mottling, increased capillary refill time—cardiogenic shock, septic • Echocardiography—bubble echocardiography, pulmonary embolism	A quick assessment of circulatory system by echocardiography is vital. It will rule out cardiac causes for hypoxia
8.	Central nervous system	• Assessment of sensorium—agitation is common in a hypoxic patient. Comatose states can "occur" with carbon dioxide (CO_2) narcosis or hypercapnia • Can patient protect his airway? • Examination of power, tone, and reflexes—neuromuscular diseases	Either CNS pathologies could be primary cause for respiratory failure or affected due to hypoxia/hypercapnia reflected the severity of disease
9.	Gastrointestinal system	• *Distended abdomen:* Ascites, ileus, increased intra-abdominal pressure—splinting of diaphragm • Cause for hypoxia—hepatopulmonary syndromes, pancreatitis (pleural effusion)	Intra-abdominal pressure monitoring may be useful to help to titrate PEEP using transpulmonary pressure

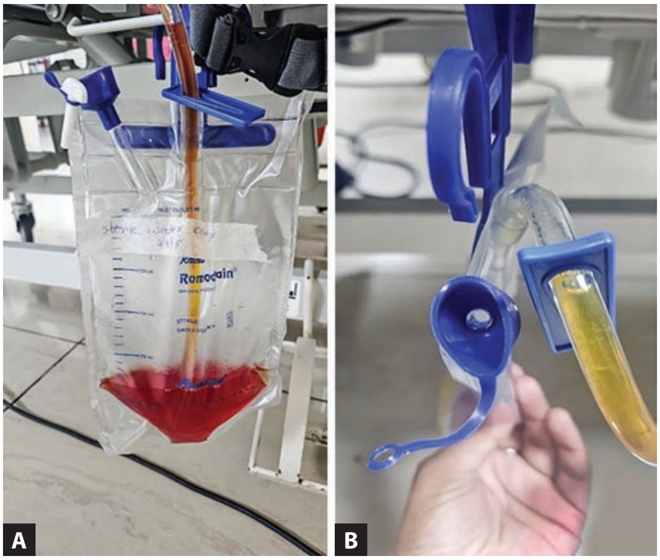
(ARDS: acute respiratory distress syndrome; CNS: central nervous system; COPD: chronic obstructive pulmonary disease; HIV: human immunodeficiency virus; IAP: intra-abdominal pressure; ICD: intercostal drain; OSA: obstructive sleep apnea; PEEP: positive end-expiratory pressure; SLE: systemic lupus erythematosus; SVC: superior vena cava)

CLINICAL EXAMINATION

General Physical Examination

- *Appearance:* Severely dyspneic patients may be orthopneic, sitting upright, or in a tripod position especially in patients with acute exacerbation of chronic obstructive pulmonary disease (COPD).
- *Nutritional status*: Is the patient obese or emaciated? Make a mental note of body mass index (BMI) on the nursing chart. Emaciation in a respiratory illness may be associated with tuberculosis (TB), malignancy, advanced COPD, or human immunodeficiency virus (HIV)-associated illness.
- *Patient's vitals:*
  - Heart rate
  - Respiratory rate

  } Patients who are hypoxic usually have associated tachypnea/tachycardia. However, absence of these does not rule out hypoxia.
  - *Blood pressure:* Patients with hypotension may have accompanying hypoxia due to poor tissue perfusion and hypoxia does not improve until the hypotension is corrected.
  - *Temperature:* Hyperthermia or hypothermia may indicate sepsis as a possible cause for hypoxia.



Figs. 1A and B: While inspecting a chest drain, ensure the underwater seal is maintained. Ensure the drainage bag or bottle is kept open to the atmosphere in case of a single drainage chamber and no clamps are placed on the tubing.

- *Oxygen saturation:* This is the 5th vital sign and quickest way to confirm hypoxemia. It is checked by placing a pulse oximeter over the patients' nailbed or earlobe.

If the patient is comfortable with no signs of respiratory distress and the pulse oximeter shows a low reading (SpO_2 <90%), it is prudent to check the pulse oximeter, check the waveform obtained, or look for spurious causes of hypoxemia such as leukocyte or platelet larceny.

Findings on general physical examination that could give a clue to cause of hypoxia are as follows:

- *Pallor:* Anemia, TB, malignancy
- *Cyanosis:* Sign of severe hypoxemia, peripheral cyanosis can be seen in other conditions with hypoxia such as Raynaud's disease, low cardiac output states
- *Clubbing:* Bronchogenic carcinoma, fibrosing alveolitis, COPD, lung abscess
- *Lymphadenopathy:* Left supraclavicular lymph node enlargement suggests a left upper lobe malignancy. Generalized lymphadenopathy is seen in autoimmune diseases, scrub typhus, leptospirosis, or viral etiology.
- *Pitting pedal edema, facial edema, sacral edema:* Congestive cardiac failure, renal insufficiency
- *Halitosis:* Suppurative lung disease
- *Koilonychia:* Iron deficiency anemia
- *Malar rash:* Systemic lupus erythematosus (SLE), mitral stenosis
- *Surgical emphysema:* Chest trauma, barotrauma, postintercostal drain (ICD) insertion
- *Skin and mucosa examination:* Check for cutaneous manifestations of autoimmune diseases such as palpable purpura, pyoderma gangrenosum, urticaria-like rashes.

Systemic Examination

Begin with a quick examination of the airway and the respiratory system.

- *Airway and breathing:* Check the patency and breathing pattern—If the patient is able to speak, we can assume the airway is patent.

In case of a partially obstructed airway, one may see signs such as a tracheal tug (downward movement of the trachea in inspiration), supraclavicular indrawing and paradoxical breathing or seesawing, i.e., the chest moves in with inspiration and outward with expiration with asynchrony between chest wall and abdomen.

- If any chest tubes or drains are present, look for any disconnections, presence of air column movement in the tubing and underwater seal. *See* **Figures 1A and B** for clinical points to observe with a chest drain.

Inspection

Fully expose the chest.

- Note the respiratory rate, usage of accessory muscles, patient posture, any inability to lie flat (orthopnea), presence of purse lip breathing, trachea position, and chest wall asymmetry.
    - Usually, the side with the pathology will demonstrate decreased movements **(Fig. 2)**.
    - Unilateral reduced movement can be seen in pneumonia, collapse, pleural effusions, pneumothorax, and rib fractures.
- Look for signs of *increased work of breathing* such as tachypnea, increased depth of respiration, and thoracic cage expansion associated with accessory muscle use (flaring of ala nasi, intercostal indrawing, contraction of the sternomastoid, and scaleni).
- Usage of accessory muscles can be seen commonly with airflow obstruction such as COPD exacerbations and acute attacks of asthma.
- In case of shallow respiration rule out recent use of narcotics, recent upper abdominal/chest surgery, or chest trauma.

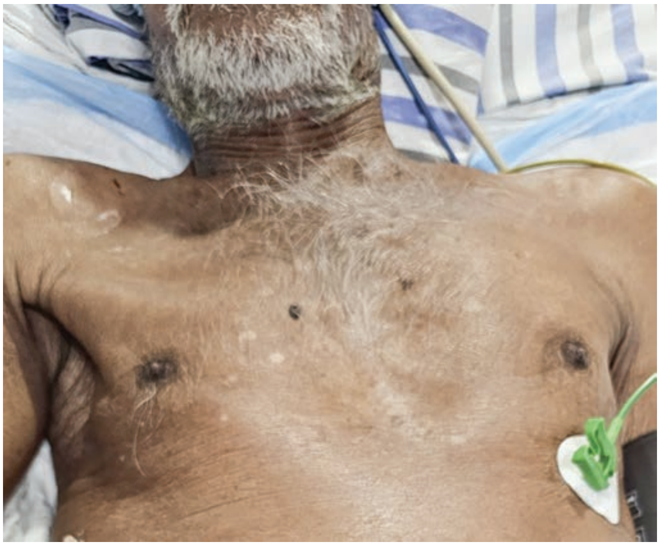


Fig. 2: Note the infraclavicular hollowing on the right side of the chest—suggestive of underlying fibrosis/collapse of right upper lobe.

TABLE 3: Chest deformities—types and clinical description.

Abnormal types of chest	***Description and causes***
Pigeon breast/ pectus carinatum	Characterized by forwarding protrusion of the sternum and adjacent costal cartilages. Seen in congenital anomalies, chronic nasopharyngeal obstruction, or rickets
Barrel-shaped chest/ emphysematous chest	The anteroposterior diameter of the chest is increased, the ribs run more horizontally, the lower ribs tend to flare upward, the sternum is arched, and the angle of Louis is prominent. This is to accommodate the lung in emphysema
Funnel/pectus excavatum	Exaggeration of the normal hollow over the lower end of the sternum. Seen in Marfan syndrome, straight back syndrome, occupational deformity as in cobbler, secondary to rickets in children
Flail chest/stove-in chest	Seen secondary to multiple rib fractures causing paradoxical movement of the chest

- *Position of the trachea:*
  - Shifted to the ipsilateral side in case of pathologies such as collapse and fibrosis.
  - Shifted to contralateral side in case of pneumothorax and pleural effusions.
- *Chest wall deformities* ***(Table 3)****:*
  - Kyphoscoliosis, poliomyelitis, and ankylosing spondylitis
  - *Barrel shaped chest:* Emphysema
  - Pectus carinatum, pectus excavatum
  - *Localized bulging:* Aortic aneurysm, empyema necessitans, surgical emphysema
- *Scars:* Previous ICD/drains, previous thoracotomy

Palpation

Localized tenderness may be present in case of any chest trauma, rib fractures.

- *Check for chest symmetry and lung expansion:* Place two hands, palms down on the patient's anterior chest with both thumbs over the anterior part of the sternum to feel for bilateral equal chest movements. Both the thumbs should move away equally with each respiration. The hand may also be placed below the patient's costal margin on either side to appreciate for symmetrical chest expansion **(Figs. 3A and B)**.
- *Tactile fremitus:* Not of much clinical significance in critically-ill patients especially for rapid determination of cause of hypoxia. The ulnar border of the examiner's hand is kept in either intercostal space with the patient saying "one" or "ninety nine". It is decreased in cases of pleural effusions and pneumothorax and increased in consolidation and lung abscess.

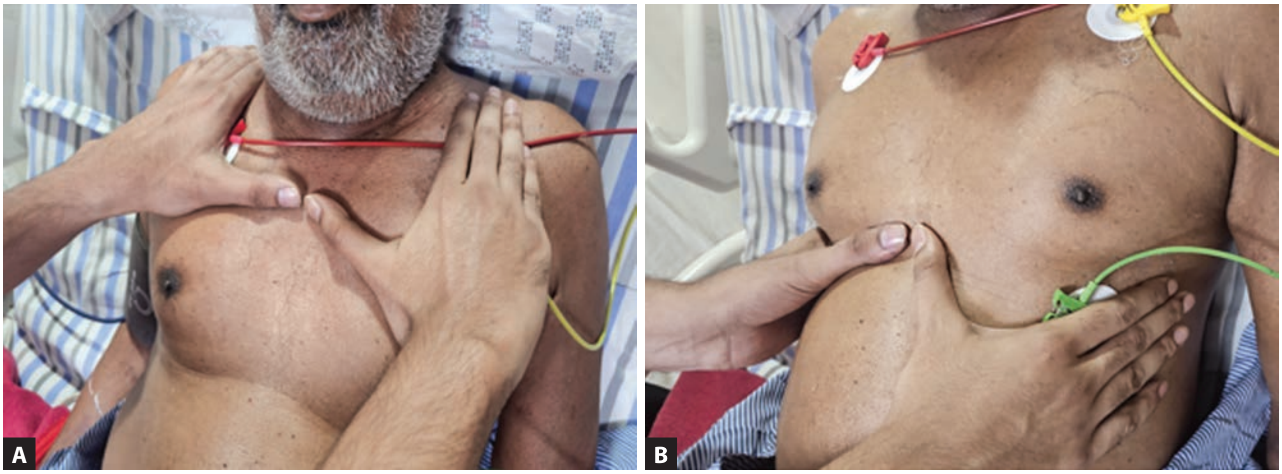
Percussion

- *Presence of dull note:* Pleural effusion, collapsed lung, dense consolidation
- *Presence of hyperresonant note:* Pneumothorax, diaphragmatic hernia with gas-filled distended stomach or intestine.

Auscultation

Normal breath sounds are generated by turbulent airflow in the upper airway, which produces a hissing sound, similar to a bronchial breath. As this sound is transmitted through the lung it is damped, higher frequencies are lost and only lower frequency (200–400 Hz) is heard. These are vesicular breath sounds. Auscultation should be done symmetrically.

- *Bilateral basilar crackles:* Seen in pulmonary edema or fluid overload (correlate with other signs such as jugular venous distention and/or lower limb edema).
- *Wheezing and rhonchi:* Seen in Asthma, obstructive lung disease, rare causes include foreign body obstruction, and anaphylaxis. Diffuse polyphonic wheeze interspersed with crackles may indicate acute exacerbation of asthma/COPD.



Figs. 3A and B: Checking for symmetrical chest expansion by placing both hands over the anterior part of the chest or at the lower costal margin. Both thumbs should move away equally with each respiration in case of symmetrical chest expansion.

- *Absent unilateral air entry:* Seen in massive pleural effusion, pneumothorax, and pneumonectomy.
- *Diminished breath sound, i.e., vesicular with reduced intensity:* Seen with pleurisy, pericarditis, weakness of respiratory muscles, and diminished excursion of the diaphragm as in ascites.

A silent chest may be appreciated in status asthmaticus.

Pleural rub is produced by rubbing of inflamed pleura. It is characterized by rubbing or creaking quality, loud in intensity, superficial in character, and increased pressure on the chest piece on the chest wall. This is audible in both phases of respiration.

Clear lung fields on auscultation in a hypoxic patient should raise suspicion of pulmonary embolism, Presence of tachycardia and a red, edematous, tender limb may help to additionally clinch the diagnosis.

The clinical examination of the respiratory system can be complemented with LUSG) at this step. See "Adjuncts to be Used for Rapid Diagnosis of Hypoxia."

Next, proceed to cardiovascular examination.

- *Clinically examine for the jugular venous pressure (JVP):* Examined with the head end of the patient raised to 45° angle to the bed and neck slightly turned to one side. Elevated JVP indicates elevated central venous pressures and may be seen in heart failure, cor pulmonale, and fluid overload states.
- Inspect the precordium and identify the apical impulse.
    - The position of the apical impulse (can be displaced or pulled toward the side of the lesion in case of lung fibrosis or collapse, and away from the side of the lesion or pushed in case of pleural effusion, empyema, and pneumothorax).
- Palpate for presence of parasternal heave or thrills.
- Auscultation for any added sounds such as S3, S4 gallop, murmurs of mitral stenosis/regurgitation, aortic stenosis/regurgitation, and flow murmurs seen in conditions such as anemia.

The clinical examination of the cardiovascular system can be complemented with screening 2D echocardiography at this step. See "Adjuncts to be Used for Rapid Diagnosis of Hypoxia."

Complete the clinical examination of a hypoxic patient with focused examination of the abdomen and the central nervous system (CNS) examination. Expose the abdomen and evaluate for any abdominal distension, tenderness on palpation, and presence of fluid thrill in case of a large ascites. CNS examination should focus on higher mental functions especially level of consciousness of the patient and examine for any evidence of neuromuscular weakness. Patients with depressed levels of consciousness and neuromuscular weakness may be hypoventilating, leading to hypoxemia due to type II respiratory failure. Patients with neuromuscular weakness may also have associated diaphragmatic weakness.

Adjuncts to be Used for Rapid Diagnosis of Hypoxia

Pulse Oximetry

This is fastest way to determine the oxygenation status of a patient in conjunction with clinical signs. The SpO_2 is the easiest parameter to monitor to see response to treatment and changes in oxygenation.

There is no fixed target SpO_2 to maintain in critically-ill patients but ranges from 88 to 94%. Oxygenation targets must be individualized as per the specific condition of the patient. It is important to use the lowest FiO_2 possible to achieve the target SpO_2.

Pulse oximetry, however, does not give information of the ventilation status of the patient. A subsequent ABG needs to be ordered to know the $PaCO_2$.

The ABGs should also be asked for if the SpO_2 readings are inaccurate or not easily obtained.

Arterial Blood Gas Analysis

It is useful to have this investigation asked for while history and clinical examination are ongoing. It gives direct information about the partial pressure of oxygen in the blood (PaO_2) which is a direct measure of oxygenation and calculation of the alveolar-arterial (A-a) gradient, and also helps to identify hypercapnia.

The A-a gradient is a measure of the difference in oxygen partial pressure between the alveoli and the arteries. It is used to evaluate the efficiency of oxygen exchange in the lungs and assess for cause of hypoxemia. It is calculated using the given formula:

$$\text{A-a gradient} = (PAO_2 - PaO_2)$$

Where:

- PAO_2 = Partial pressure of oxygen in the lungs/alveoli.
- PaO_2 = Partial pressure of oxygen in arterial blood.

PAO_2 is estimated from the alveolar gas equation as follows:

$$PAO_2 = [(Pb - PH_2O) \times FiO_2] - (PaCO_2/RQ)$$

Where:

- Pb is the barometric pressure.
- PH_2O is the partial pressure of water vapor in the alveoli.
- FiO_2 is the fraction of inspired oxygen.
- $PaCO_2$ is the partial pressure of carbon dioxide in the arterial blood.
- RQ is the respiratory quotient (typically assumed to be 0.8).

The normal A-a gradient ranges between 5 and 25 mm Hg.

A higher A-a gradient indicates a larger difference between the alveolar and arterial oxygen levels, suggesting impaired gas exchange in the lungs. It suggests a problem with oxygen diffusion or ventilation-perfusion (V/Q) mismatch, such as in conditions like pneumonia, acute respiratory distress syndrome (ARDS), pulmonary embolism, or interstitial lung diseases.

The A-a gradient will be normal in hypoventilation.

Chest X-ray

Although it may not be available rapidly, it is a useful investigation that can identify consolidation, collapse, pulmonary edema, and life-threatening problems like tension pneumothorax.

Limitation: Time bound and not useful for rapid diagnosis.

Point-of-Care Ultrasonography (POCUS)

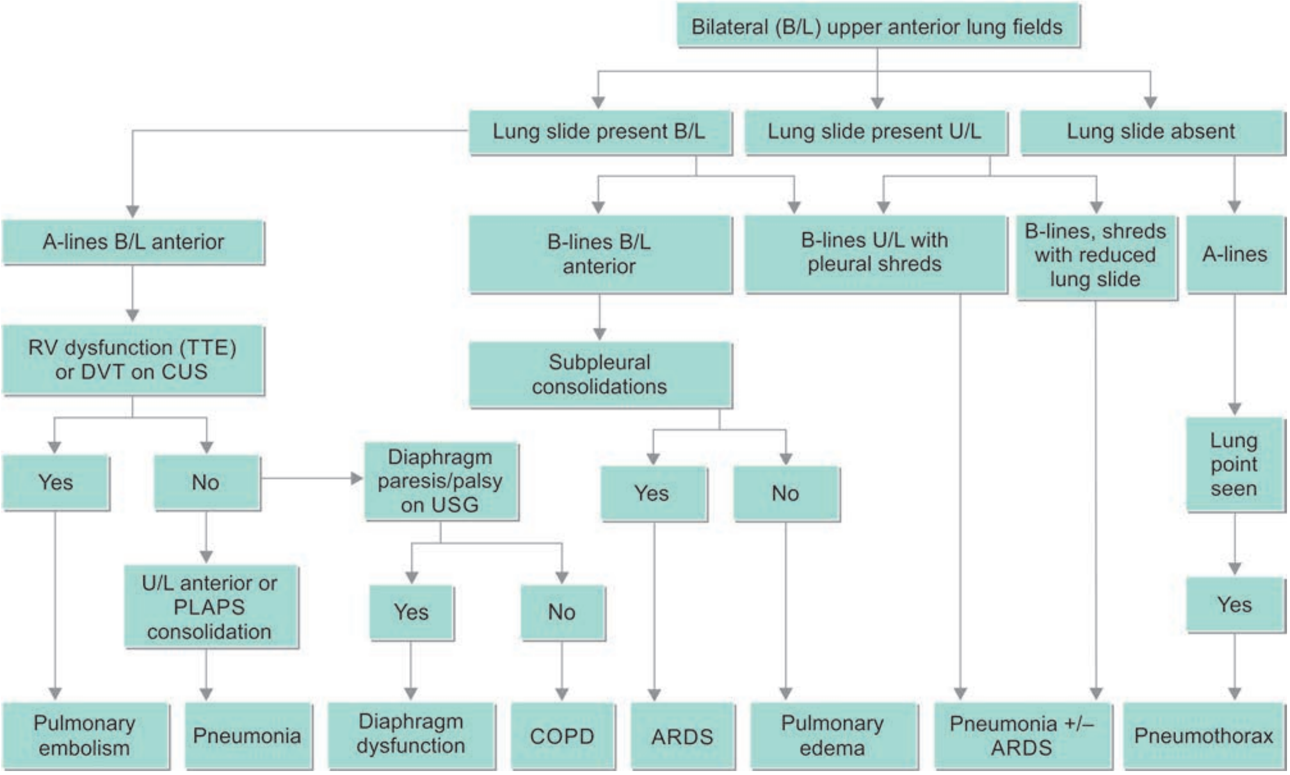
Lung ultrasonography: Valuable tool in rapid diagnosis of cause of hypoxia. Various protocols have been developed for patients with respiratory failure but the most widely used is the BLUE (bedside lung USG in emergency) protocol by Daniel Lichenstein et al. This algorithm is directed at identifying causes of acute respiratory failure in under 3 minutes. **Flowchart 1** shows a modified BLUE protocol algorithm.

Table 4 shows classical signs of LUSG with its interpretation.

Clinical applications of lung USG in hypoxemia:

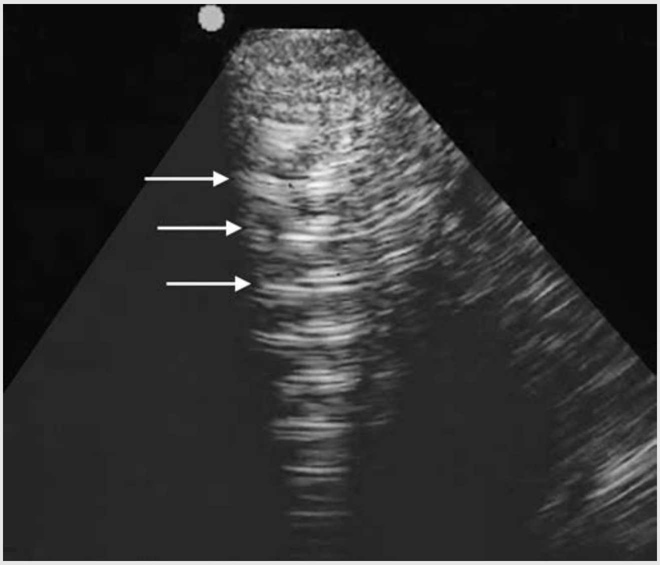
- *Acute respiratory distress syndrome:* LUSG can differentiate ARDS from cardiogenic pulmonary edema. In ARDS, multiple diffuse B-lines, consolidations, and spared areas are common, whereas cardiogenic pulmonary edema typically presents with bilateral, symmetrical B-lines without consolidations.
- *Pneumonia:* LUSG is highly sensitive and specific for diagnosing pneumonia, with findings including subpleural consolidations, dynamic air bronchograms, and localized B-lines. It is particularly useful in detecting pneumonia in critically-ill patients where transport for radiographic imaging is challenging.
- *Pulmonary embolism:* While LUSG cannot directly visualize pulmonary emboli, it can identify secondary signs such as pleural-based consolidations and infarctions. Additionally, the absence of other causes of hypoxemia (e.g., pneumothorax, pleural effusion) can indirectly suggest pulmonary embolism in the appropriate clinical context.
- *Heart failure:* In heart failure, LUSG reveals bilateral B-lines (interstitial syndrome) and pleural effusions. These findings, combined with clinical and

Flowchart 1: Diagnosing cause of hypoxemia using lung ultrasonography.


Bilateral (B/L) upper anterior lung fields
Lung slide present B/L
Lung slide present U/L
Lung slide absent
A-lines B/L anterior
B-lines B/L anterior
B-lines U/L with pleural shreds
B-lines, shreds with reduced lung slide
A-lines
RV dysfunction (TTE) or DVT on CUS
Subpleural consolidations
Lung point seen
Yes
No
Diaphragm paresis/palsy on USG
Yes
No
U/L anterior or PLAPS consolidation
Yes
No
Yes
Pulmonary embolism
Pneumonia
Diaphragm dysfunction
COPD
ARDS
Pulmonary edema
Pneumonia +/– ARDS
Pneumothorax

(ARDS: acute respiratory distress syndrome; COPD: chronic obstructive pulmonary; CUS: compression ultrasonography; DVT: deep vein thrombosis; PLAPS: posterolateral and pleural syndrome; RV: right ventricle; TTE: transthoracic echocardiography; USG: ultrasonography; U/L: unilateral)

TABLE 4: Classical findings on lung ultrasonography.

Normal lung		
A-lines	• Horizontal, repetitive reverberation artifacts parallel to the pleural line, indicative of normal aeration • The white arrows show the reverberation artifacts from the pleural line or the A-lines	

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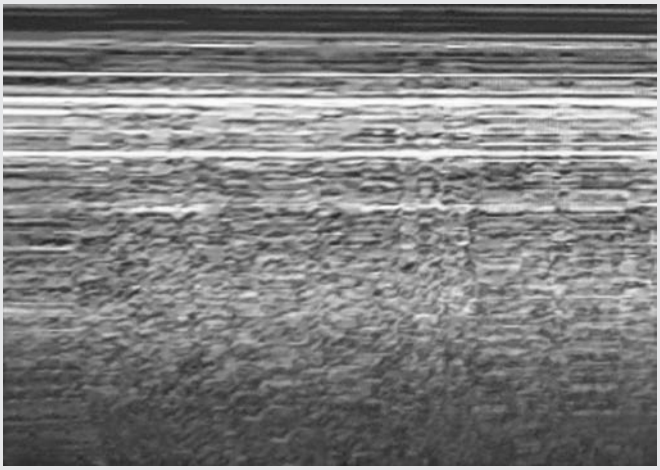
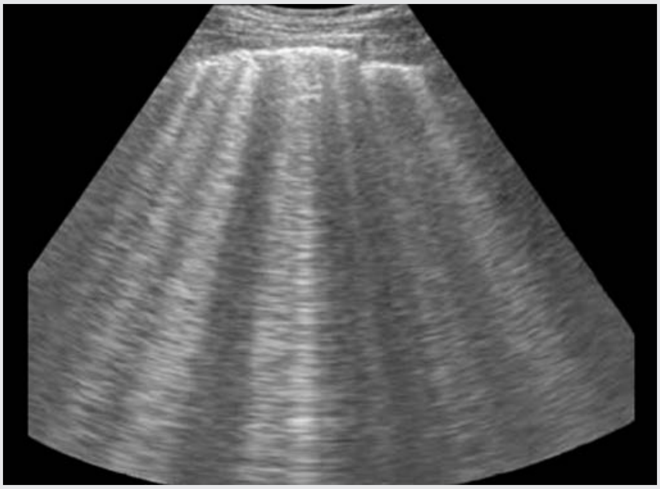
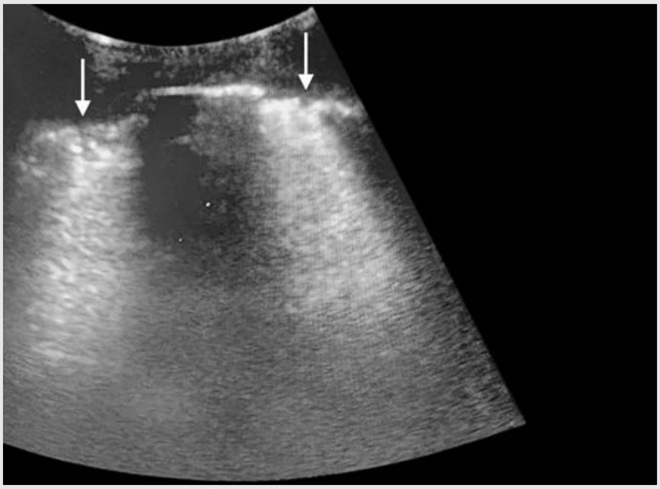
Normal lung		
Lung sliding	• Shimmering motion of the pleural line during respiration, indicating normal lung expansion and aeration • Use of the M mode or motion mode can reveal the "seashore sign" in case of normal lung sliding (see image on right)	
Pathological findings		
B-lines	Vertical, comet-tail artifacts originating from the pleural line, moving with lung sliding. Multiple B-lines suggest interstitial syndrome, often seen in conditions such as pulmonary edema or interstitial lung disease	
Pleural shreds	The pleural line becomes fragmented or irregular (see arrow on right), showing multiple small interruptions. B-lines could be seen arising from shredded pleura. They are suggestive of nontranslobar consolidations, a typical finding seen in ARDS, pulmonary contusions, etc.	

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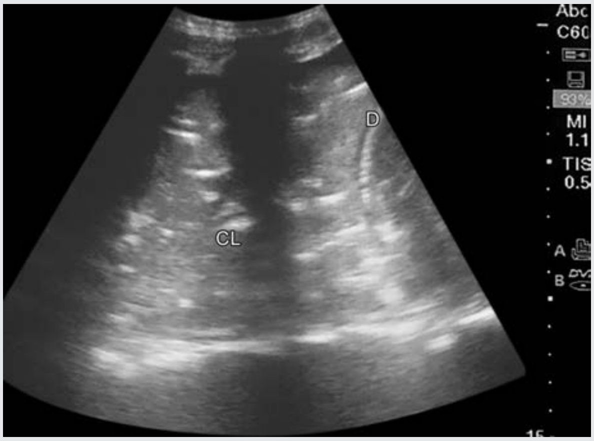
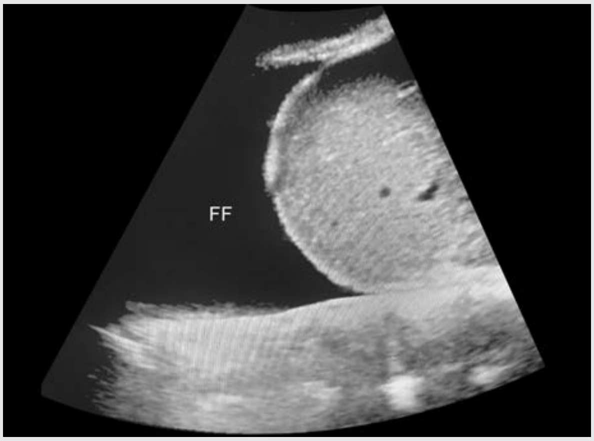
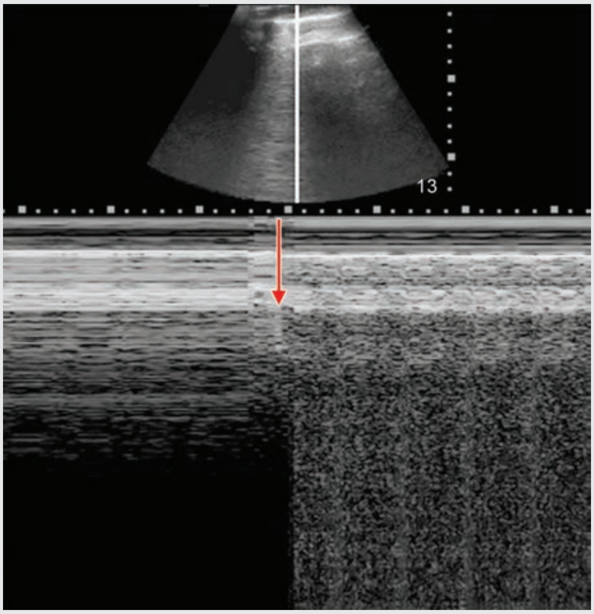
Pathological findings		
Tissue sign	- Hypoechoic or tissue-like areas within the lung parenchyma, representing alveolar filling processes such as pneumonia or atelectasis - Suggests a translobar consolidation (marked CL in the image, D—diaphragm)	
Pleural effusion	Anechoic or hypoechoic fluid collection in the pleural space, typically seen in heart failure, infection, or malignancy	 FF = free fluid, seen in pleural effusion
Pneumothorax	Absence of lung sliding and the presence of the "lung point" (the point where the normal lung and pneumothorax meet), identified by a sudden change from lung sliding to no sliding (see red arrow which marks the lung point)	

(ARDS: acute respiratory distress syndrome; CL: consolidation) echocardiographic data, assist in the rapid diagnosis and management of acute heart failure.

- *Pneumothorax:* LUSG is superior to chest radiography in diagnosing pneumothorax. The absence of lung sliding, presence of A-lines, and detection of the lung point are diagnostic hallmarks.
- *Interstitial lung disease:* Chronic hypoxemia due to interstitial lung diseases can be evaluated using LUSG, which shows multiple bilateral B-lines, thickened pleural lines, and subpleural consolidations.

Screening 2D echocardiography: Look for changes of left ventricular systolic/diastolic dysfunction, right atrial/right ventricular dilatation, right ventricular dysfunction, regional wall motion abnormalities, pericardial effusions, cardiac tamponade, patent foramen ovale, and presence of intracardiac vegetations/clots.

Compression ultrasonography: A 2 point or 3 point CUS can be performed to rule out presence of deep vein thrombosis (DVT). Inability to compress the deep veins on USG, echogenic material noted within the venous lumen or absent flow on duplex venous USG is highly suggestive of DVT. A positive CUS in conjunction with A-profile on LUSG in a hypoxic tachycardic patient should strongly raise the suspicion of acute pulmonary thromboembolism.

USG of the airway and the diaphragm: Airway USG can help to confirm endotracheal intubation, rule out esophageal intubation, and endobronchial intubation as causes of hypoxia. Diaphragm USG can help rule out any diaphragmatic paresis and diaphragmatic dysfunction as a cause of respiratory failure.

Electrocardiography

Electrocardiogram (ECG) changes are suggestive of ischemia, myocardial infarction, arrhythmias, and bundle branch blocks that could indicate cardiovascular causes for hypoxemia.

The patient has worsening hypoxemia and respiratory distress. You decide to intubate the patient and put him on volume controlled mode tidal volume (Vt) 410 mL, respiratory rate—28 bpm, FiO_2—0.8, and positive end-expiratory pressure (PEEP)—8 cmH_2O. 4 hours later you manage to get the FiO_2 to 0.5 and within half an hour, the nurse calls you stating that the patient is desaturating. How would you proceed?

Perform a comprehensive evaluation to assess the cause which should include the following:

- Clinical assessment (described previously)
- Monitoring ventilator graphics
- Use of ABG and capnography
- *Bedside imaging:* LUSG and 2D ECHO, CUS, and chest X-ray.

One needs to identify if the problem causing hypoxia is arising from the patient or the ventilator equipment. If the ventilator malfunctioning is suspected, first disconnect the patient from the ventilator and ventilate the patient with 100% oxygen using an Ambu bag.

An increased resistance to ventilate may suggest an increased resistance or decreased lung/chest wall compliance. Disconnection from the ventilator can also help alleviate issues of dynamic hyperinflation in case of intrinsic PEEP.

Use of capnography can help in identifying any tube misplacement/accidental extubation.

See **Box 1** for the possible causes of hypoxia in a mechanically ventilated patient.

BOX 1: Causes of hypoxia in a ventilated patient.

Common causes of respiratory distress in a mechanically ventilated patient.

Equipment related:
- Endotracheal tube (ETT) block
- ETT migration
- Tube displacement
- Cuff leak
- Ventilator malfunction
- Changes/errors in ventilator settings

Patient factors:
- Acute cardiogenic pulmonary edema
- Aspiration
- Pneumothorax
- Inadequate sedation
- Decreased chest wall compliance due to gross ascites or large pleural effusions

ROLE OF VENTILATOR GRAPHICS

Ventilator waveforms can give much information about the cause of hypoxia. Check the peak airway pressures (Ppeak) and plateau pressures (Pplat) if the patient is on a volume controlled mode of ventilation (VCV). To obtain the Pplat on VCV, press the "inspiratory hold" for 3–5 seconds.

A rise in Ppeak alone indicates an issue of increased airway resistance such as endotracheal tube (ETT) obstruction, bronchospasm, etc. While an increase in both Ppeak and Pplat would highlight an issue of pulmonary or chest wall compliance. A delta Ppeak and Pplat value (i.e., the difference between Ppeak and Pplat) when both are elevated can help to identify dual issues of increased airway resistance with decreased lung/chest wall compliance. Ppeak – Pplat of greater than 10 cmH_2O can indicate higher airway resistance **(Fig. 4)**.

Table 5 gives the possible etiologies, assessment, and treatment for possible causes of hypoxia in mechanically ventilated patient.

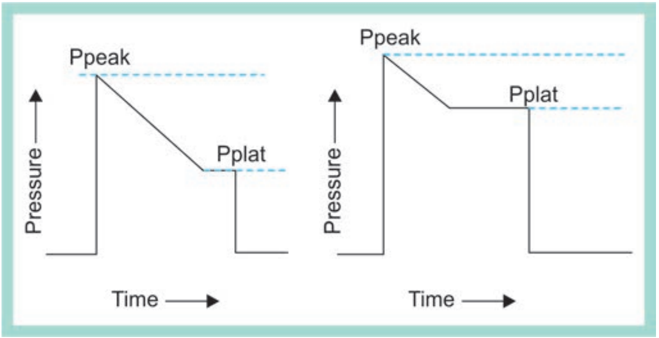


Fig. 4: Ventilator graphics in volume controlled ventilation. In the image on the left, there is an increase in Ppeak with no change in Pplat and increase in delta Ppeak – Pplat suggestive of increased resistance as seen with bronchospasm, obstruction of endotracheal tubes, etc.

The image on the right is suggestive of increase in both Ppeak and Pplat suggestive of decreased lung/chest wall compliance as seen with pneumothorax, pulmonary edema, ARDS, large pleural effusions, etc. (Ppeak: peak airway pressures; Pplat: plateau pressure)

TABLE 5: Possible etiologies, diagnosis, and management of hypoxia in a mechanically ventilated patient.

	Clinical findings	***Ventilator graphics***	***Bedside testing***	***Treatment***
Issues with ventilator				
Incorrect settings	Inappropriate FiO_2/PEEP settings/set tidal volume/respiratory rate/alarm limits	Inappropriate FiO_2/PEEP settings/set tidal volume/respiratory rate/alarm limits	Disconnect ventilator ad ventilate with Ambu bag and 100% O_2. If that resolves the issue, check the ventilator settings	• Make changes as needed to improve oxygenation • Change mode to controlled modes If needed • Change the ventilator if found faulty
Airway issues				
ETT obstruction	• Thick secretions in ETT • Mucus plugging • Kinking in the ETT • Patient biting the tube • Foreign body in ETT/airways	• Increased peak airway pressures with increased delta Ppeak – Pplat • Look at the flow volume loops and scalars for the "seasaw" pattern seen with presence of secretions in the circuit **(Fig. 5A)**	• Auscultate the patient and look for any reduced air entry, added sounds • Perform a suction and check for resistance to passing the suction catheter	• Treat the underlying cause of the obstruction, e.g., suctioning thick secretions • Place bite block, use of sedation/paralytics if needed • Change the ETT if obstruction still persists • Bronchoscopy if needed
Leak, cuff deflation, or rupture	Audible leak	Difference in inhaled and exhaled tidal volume on monitor and flow volume loops **(Fig. 5B)**	Check closely for any leaks in the circuit/HME filter	• May warrant change of ETT/circuit, HME filter • Use capnography to confirm tube position
Bronchospasm	Exacerbation of COPD, asthma anaphylaxis due to increased secretions	Increased peak airway pressures with increased delta Ppeak – Pplat	Auscultate for presence of rhonchi	Bronchodilators, IV magnesium sulfate, treat any underlying infective cause
ETT displacement/endobronchial intubation	Cuff leak in case or cephalad migration, decreased air entry on left in case of endobronchial intubation	Increased peak airway pressures and Pplat	LUSG or airway USG, direct laryngoscopy, CXR	• Reposition the tube/reintubate as necessary • Confirm tube position with capnography and CXR

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	Clinical findings	***Ventilator graphics***	***Bedside testing***	***Treatment***
Pulmonary parenchymal pathologies				
Atelectasis	Reduced air entry and unilateral chest movement on affected side, tracheal shift to affected side	Increased Ppeak and Pplat with no increase in delta Ppeak – Pplat	• LUSG—tissue sign with static air bronchogram and lung pulse, reduced lung sliding • CXR may also show affected lobes	Chest physiotherapy, recruitment maneuvers, bronchoscopy if needed, pleurocentesis in case of collapse due to large underlying effusions
Pneumonia	Fever, change in nature of secretions, crepitations on auscultation of affected side	Increased Ppeak and Pplat with no increase in delta Ppeak – Pplat	CXR and LUSG may reveal features of consolidation	Adequate antimicrobial therapy
Pulmonary embolism (PE)	• Pain and swelling of lower extremity • Fever may be seen	May be normal	LUSG may show a normal A-profile, echocardiography may reveal right atrial/ventricular enlargement with right ventricular dysfunction. Compression USG to rule out a DVT	• Anticoagulation • Thrombolysis in case of massive PE with no contraindications
Pulmonary edema	Bilateral fine crepitations on auscultation, Presence of raised JVP, murmurs, pink frothy secretions in ETT, positive cumulative fluid balance	Increased Ppeak and Pplat with no increase in delta Ppeak – Pplat	LUSG may reveal a B-profile. Echocardiography may show left ventricular (LV) systolic/diastolic dysfunction with elevated filling pressures. ECG to rule out ischemia, arrhythmias	• Diuresis • Ultrafiltration • Treat the underlying cause
Dynamic hyperinflation	Suspect in patients ventilated for obstructive airway disease, high respiratory rates. May be associated with hypotension	Inadequate return of expiratory flow to baseline before the next ventilator-delivered breath **(Fig. 5C)**		• Disconnect the ventilator transiently • Change ventilator settings allowing adequate time for lung emptying (reduce respiratory rate, increase time for exhalation) • Correction of bronchospasm
Pleural causes				
Pneumothorax	• Reduced breath sounds on affected side, tracheal deviation if tension is present • Percussion of affected side may show a hyperresonant note	• Increased Ppeak with increased delta Ppeak – Pplat • Reduced tidal volume if associated with a bronchopleural fistula	LUSG may reveal absent lung sliding with lung point (pathognomonic of pneumothorax)	Rapid decompression of pneumothorax with a thoracostomy tube

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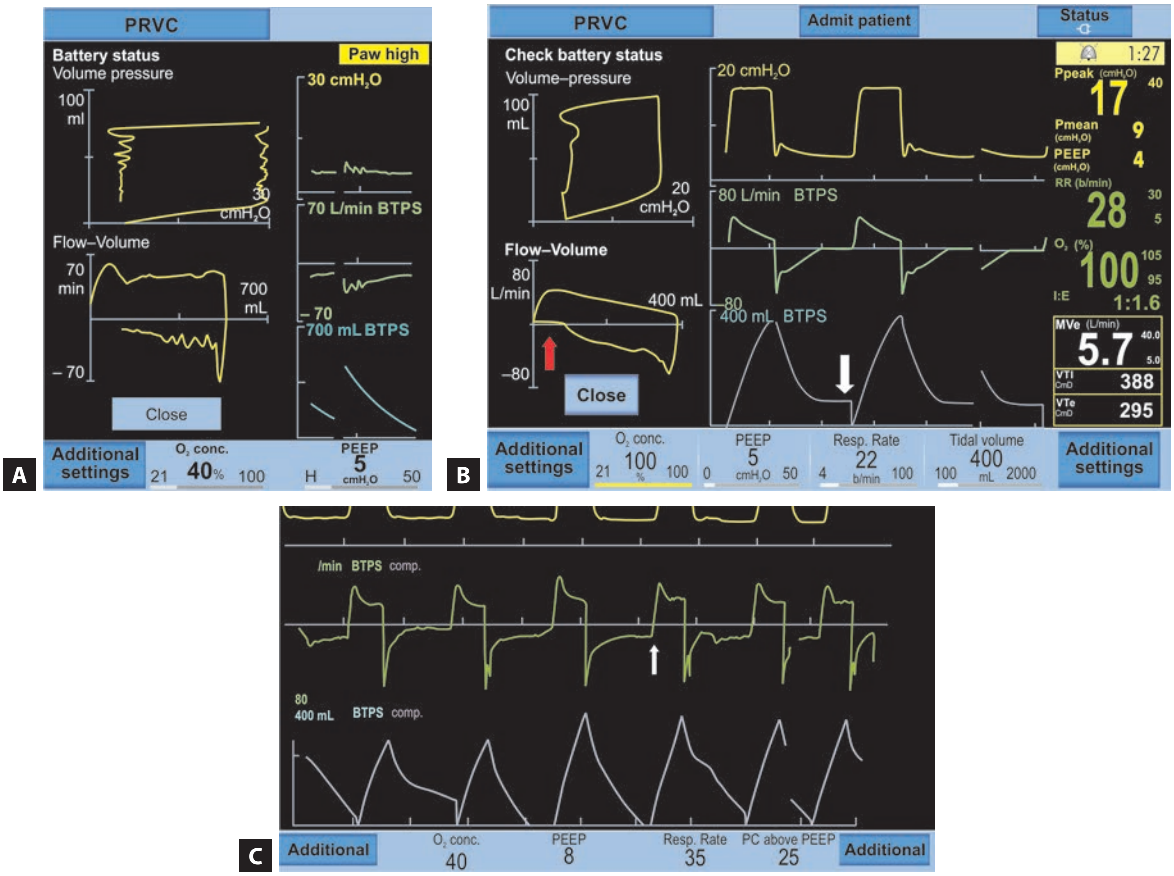
	Clinical findings	***Ventilator graphics***	***Bedside testing***	***Treatment***
Extrapulmonary issues				
Increase in intra-abdominal pressures, e.g., ascites, hollow viscous perforation, abdominal compartment syndrome	Abdominal distension, pain, tenderness, feed intolerance	• Increase in both Ppeak and Pplat in VCV • On PCV, decrease in tidal volume, rapid return of expiratory flow toward baseline may be seen	Use of clinical examination aided with USG and intra-abdominal pressure monitoring if needed	• Large volume paracentesis • Treat the underlying cause
Fever, pain, agitation, delirium	More often a diagnosis of exclusion			Treat the underlying cause

(COPD: chronic obstructive pulmonary disease; CXR: chest X-ray; DVT: deep vein thrombosis; ECG: electrocardiogram; ETT: endotracheal tube; FiO_2: fraction of inspired oxygen; HME: heat and moisture exchanger; IV: intravenous; JVP: jugular venous pulse; LUSG: lung ultrasonography; PCV: pressure controlled ventilation; PEEP: positive end expiratory pressure; Ppeak: peak airway pressure; Pplat: plateau pressures; USG: ultrasonography; VCV: volume controlled ventilation)


A
PRVC
Battery status
Volume pressure
Paw high
100 ml
30 cmH2O
Flow–Volume
70 min
700 mL
– 70
70 L/min BTPS
700 mL BTPS
Close
Additional settings
O2 conc.
21 40% 100
PEEP
5
H cmH2O 50
B
PRVC
Admit patient
Status
Check battery status
Volume–pressure
100 mL
20 cmH2O
20 cmH2O
80 L/min BTPS
Flow–Volume
80 L/min
400 mL
–80
400 mL BTPS
Close
1:27
Ppeak (cmH2O) 17 40
Pmean (cmH2O) 9
PEEP (cmH2O) 4
RR (b/min) 28 30 5
O2 (%) 100 105 95
I:E 1:1.6
MVe (L/min) 5.7 40.0 5.0
VTi CmD 388
VTe CmD 295
Additional settings
O2 conc. 21 100 % 100
PEEP 0 5 cmH2O 50
Resp. Rate 4 22 b/min 100
Tidal volume 100 400 mL 2000
Additional settings
C
/min BTPS comp.
80
400 mL BTPS comp.
Additional
O2 conc. 40
PEEP 8
Resp. Rate 35
PC above PEEP 25
Additional

Figs. 5A to C: (A) The "seasaw pattern" in the flow volume loops and pressure volume loops seen with presence of secretions in the circuit; (B) Ventilator graphics showing airflow leak (see white arrow). Note the expiratory volume does not touch the baseline at 0 indicating a leak of close to 100 mL, which can also be picked up in the flow volume scalar (red arrow). The difference between inspired and expired tidal volumes (VTi and VTe) also indicates the amount if leak present; (C) Ventilator screen showing a patient with presence of expiratory flow limitation in the flow time scalar (white arrow) (white arrow).

SUMMARY

- In an unstable hypoxic patient, always tend to the patient's airway, breathing, and circulation first. In a stable patient with hypoxia, start with oxygen supplementation and proceed systematically with history taking and clinical examination.
- Pulse oximetry, ABG, and POCUS are the best adjuncts for rapid diagnosis of cause of hypoxemia.
- In an intubated patient, look at the ventilator graphics to identify causes of hypoxemia such as increased resistance, decreased compliance, and dynamic hyperinflation.
- While there are several therapies to improve patient's oxygenation and ventilation, it is important to thoroughly investigate and treat the underlying cause in any patient with acute hypoxia.
- Consider a broad differential diagnosis in cases of hypoxia which remain refractory to standard treatment.

SUGGESTED READING

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Chapter

16

How will I Examine a Patient with an Acute Alteration in Sensorium?

Amarja Ashok Havaldar

ABSTRACT

Examining a patient presenting with an acute alteration in sensorium poses a significant challenge to intensivists. To identify whether alteration of the sensorium is primarily due to central nervous system disorder or as an effect of metabolic derangements is vital. Given the difficulties due to limited neurological examination, the stepwise approach helps in integrating the information obtained by history, clinical examination, and imaging. The chapter unfolds with the case scenario of a hypertensive patient presenting with the altered sensorium to the emergency room. As we know taking history is important, but make sure initial stabilization is done by examining airway, breathing, and circulation.

INTRODUCTION

Neurological examination in critically ill is challenging as the patients are on sedation and mechanical ventilation, which preclude detailed neurological evaluation.

Stepwise approach for neurological evaluation is as follows:

- Detailed history of presenting illness
- History of associated comorbidities and medications
- *Focused neurological evaluation:*
  - Levels of consciousness
  - Higher mental functions
  - Cranial nerves
  - Motor and sensory examination
  - Extrapyramidal evaluation

CASE SCENARIO

A 45-year-old male known hypertensive, noncompliant with medications presented with sudden onset of altered sensorium. On evaluation at emergency, he was drowsy, not arousable with no eye opening to painful stimulus with abnormal flexion of right upper limb and was making incomprehensible sounds. He was intubated in view of airway protection. Neuroimaging revealed intraparenchymal bleed. He was shifted to intensive care unit (ICU) for further management.

History and Symptomatology

The focused history of presenting illness with chronological details including comorbidities and ongoing medications should be noted. For example, diabetic patient presented with fever with dysuria develops decreased urine output and altered sensorium after 2 days; this raises the possibility of urosepsis with acute kidney injury (AKI) and uremic/septic encephalopathy leading to the altered sensorium.

The common etiologies resulting in the altered sensorium due to neurological causes can be triaged as structural and nonstructural causes.

The importance of each comorbidity has been depicted in **Table 1**.

Associated Symptoms

- Fever with headache—meningitis and meningo-encephalitis
- Seizure—cerebral venous thrombosis (CVT), subarachnoid hemorrhage (SAH), intracranial bleed, stroke, and hypertensive encephalopathy
- Headache—CVT
- Loss of consciousness—syncope and trauma

Systemic Disorders

Altered sensorium could be a manifestation of systemic disorders:

- Septic encephalopathy
- Metabolic encephalopathy—electrolyte disturbances and Wernicke's encephalopathy
- Hepatic encephalopathy

TABLE 1: Importance of comorbidity.

Comorbidities	*Presentation*
Hypertension	• Hypertensive emergency • Hypertensive bleed • Subarachnoid hemorrhage (SAH)
Diabetes mellitus	• Hypo- or hyperglycemia • Hyperosmolar hyperglycemic syndrome • Neuroglycopenic syndrome
Kidney disease	• Uremic encephalopathy • Posterior reversible encephalopathy syndrome (PRES)
Chronic obstructive pulmonary disease (COPD)	Hypercapnic respiratory failure
Liver disease	Hepatic encephalopathy
Endocrine disorders	Thyroid disorders, e.g., myxedema coma
History of trauma	Evaluation as per the advanced trauma life support (ATLS) protocol

- Uremic encephalopathy
- Drug intoxication

EXAMINATION

Purpose of the Examination

- To identify the neurological disease and localize the site of the lesion.
- To exclude any systemic diseases known to cause altered sensorium.

Initial Management

Initial management remains similar to any critically ill patient such as airway, breathing, circulation, disability, and exposure.

- *Airway:* Management of the airway includes identifying threatened airway, manual inline stabilization in case of cervical spine injuries or for patients presenting with dangerous mechanism of trauma, use of airway adjuncts to maintain airway patency, or invasive ventilator support to correct the severe hypoxia. Glasgow Coma Scale (GCS) score of <8 warrants intubation and mechanical ventilation to protect the airway.
- *Breathing:* Invasive ventilation helps to correct hypoxia and to achieve better control over breathing. Changes in CO_2 adversely affect neurophysiology. Hypercapnia can lead to a marked elevation in the intracranial pressure, whereas hypocapnia can lead to reduced cerebral perfusion and seizure.
- *Circulation:* The presence of hypotension resulting in syncope and acute decline in sensorium can be seen in patients with arrhythmias causing reduced perfusion. However, the presence of hypertension raises the possibility of raised intracranial tension, which may be associated with bradycardia, Cushing's reflex. Patients can have autonomic dysfunction leading to marked fluctuations in hemodynamics.
- *Disability:* This is aimed to check for pupillary asymmetry, motor posturing, or lateralizing signs. It can be evaluated using the GCS score and AVPU (Alert, Verbal, Pain, Unresponsive) score or FOUR score, followed by a quick neurological assessment.
- *Exposure:* To look for associated injuries in trauma, signs of needle pricks in intravenous drug abusers, or to look for rash or petechiae in suspected meningococcemia.

General Examination

General examination and decubitus will aid in determining the cause of neurological dysfunction.

- *Pallor*—syncopal episode/severe blood loss, may be seen in brainstem vascular insufficiency
- *Sweating*—activation of the autonomic nervous system—hypoglycemia
- *Jaundice*—suggests primary liver disease or drug toxicity
- *Cherry red color*—carbon monoxide poisoning
- *Corrosion of lips and mouth*—indicates poisoning
- *Needle marks*—drug abuse/medication
- *Emaciation*—advanced malignancy
- *Involuntary movements:*
    - Convulsions—if limited to one part of the body—indicates structural disease of the opposite hemisphere. If bilateral—suggests diffuse cerebral disorder.
    - Myoclonus—multifocal movement involving large muscle groups
    - Chorea—involuntary and nonrhythmic movements
    - Tremors—rhythmic movements which can occur at rest or while doing a certain task. Intentional tremors are seen in cerebellar dysfunction. Tremors at rest are seen in parkinsonism.
    - Dystonia—twisting movement with abnormal posturing

- Asterixis—it is a flapping movement of the muscles. It is seen in hepatic encephalopathy, renal failure with uremic encephalopathy, and type II respiratory failure.

Further General Examination

Pulse and Blood Pressure

The presence of elevated blood pressure and bradycardia is indicative of Cushing's reflex.

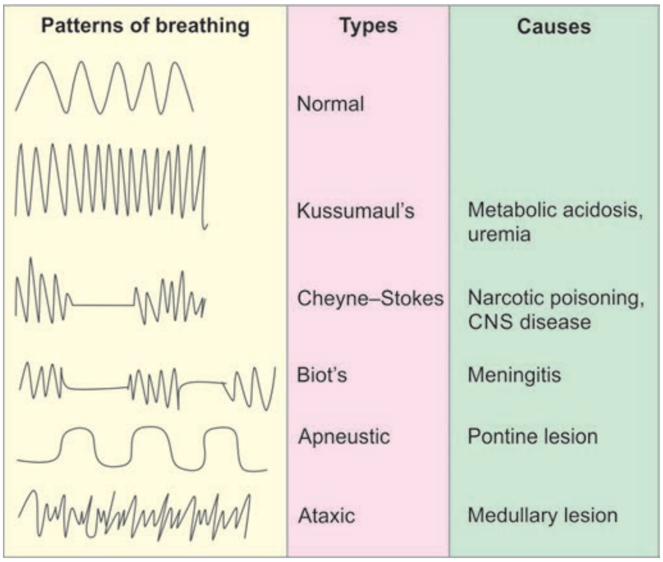
Respiration

Bradypnea is seen in drug intoxication due to sedatives.

Respiratory patterns are useful in localizing the source of an acute decline in the sensorium.

Types of respiratory patterns:

- Kussmaul's—metabolic disturbances, diabetic ketoacidosis, and uremia
- *Cheyne-Stokes breathing*—hyperventilation with apneic episodes. This is seen in lesions involving diencephalon.
- *Biot's breathing*—breathing pattern which is shallow or deep and associated with periods of apnea **(Fig. 1)**.
- *Apneustic breathing*—inspiration followed by a pause. This is seen in pontine lesions.
- *Ataxic breathing*—irregular breathing pattern with shallow and deep breaths. It is observed in medullary lesions.



Patterns of breathing	Types	Causes
	Normal	
	Kussmaul's	Metabolic acidosis, uremia
	Cheyne–Stokes	Narcotic poisoning, CNS disease
	Biot's	Meningitis
	Apneustic	Pontine lesion
	Ataxic	Medullary lesion

Fig. 1: Breathing patterns and causes.
(CNS: central nervous system)

Temperature

Hyperthermia is seen in sepsis or central fever. The various causes of hyperthermia are SAH, hypothalamic dysfunction, and pontine hemorrhage. It is also observed in heatstroke, neuroleptic malignant syndrome, malignant hyperthermia, or serotonin syndrome.

Hypothermia is generally seen due to severe sepsis, hypoglycemia, and hypothyroidism.

Neurological Examination

Consciousness

Consciousness is an awareness of the self and the surroundings. Consciousness is comprised of two components—arousal and awareness. Arousal or sleep-wakefulness is a function of ascending reticular activating system. Awareness is determined by the activity of the cerebral cortex. Both are required to determine the consciousness level. The major stages include minimally reduced consciousness, drowsiness, stupor, and coma.

Examination based on degree of altered sensorium:

Minimally reduced consciousness: This is characterized by clouding of consciousness and minimally affected arousal and awareness.

Drowsiness or lethargy: It resembles normal sleepiness, with complete wakefulness on stimulation. Generally, a routine neurological examination can be done.

Stupor: Patient arousal is noted on vigorous stimulation; once stimulation ends, the patient will return to the original state.

Coma: The patient is unconscious and difficult to arouse with stimulation.

What Happens in Locked-in Syndrome?

Locked-in syndrome occurs due to the lesion involving the ventral pons. Patient will have quadriparesis with preserved consciousness and responds with vertical eye movements and blinking.

Types of locked-in syndrome are described as follows:

- *Classical*—intact cognition and vertical gaze movement
- *Incomplete*—presence of eye movements with the additional voluntary movements
- *Total*—complete paralysis

Examination of other associated altered sensorium states

Confusion/disorientation: Generally, the confused patient will be alert and cooperative, however, may have abnormal comprehension. Hence, testing should include the following three domains:

- Time—to ask for the day, date, month, season, and year
- Place—to ask where the patient is and what country/town/building/floor/room the patient is in
- Person—to ask for the patient's identity—age, work, and the identity of the relatives.

Delirium: It is acutely disturbed state of mind which is characterized by restlessness, illusions, and incoherence with associated motor activity and physical exhaustion. The components of delirium include the presence of fluctuating mental state with inattention, altered level of consciousness, and disorganized thinking. The symptoms show diurnal variation where symptoms are worst in the evening. The types of delirium include hyperactive, hypoactive, and mixed.

The gold standard for delirium evaluation is the Diagnostic and Statistical Manual of Mental Disorders-4 (DSM IV). Two validated scales for the critically ill patients are—Confusion Assessment Method ICU (CAM-ICU) and the Intensive Care Delirium Screening Checklist (ICDSC).

Catatonia: It is a psychotic state in which the patient lies immobile and unresponsive.

In functional disorders or malingering, neurological findings will be inconsistent.

Rapid withdrawal from the painful stimuli and resistance to eye opening are the peculiar features.

Glasgow coma scale: Glasgow Coma Scale includes three particular aspects of behavioral response as described earlier **(Table 2)**.

Glasgow Coma Scale can be used to classify head injuries into mild (13–15), moderate (9–12), and severe (<8)

Glasgow Coma Scale <8 warrants securing the airway.

There are certain limitations in using GCS, as it takes into account the best response for eye, verbal, and motor components. In patients with hemiparesis or monoparesis, the motor component can remain 6, and will not give an idea about associated motor deficits. Similarly, patients with a GCS of 15 can have impaired airway protection due to stroke involving the brainstem.

Another method for evaluation of GCS is (full outline of consciousness) FOUR score, which has four components, eye, motor response, brainstem reflexes, and respiratory pattern with a maximum score of 16. The FOUR score is a better predictor of mortality in critically ill patients.

TABLE 2: Glasgow Coma Scale.

Eye opening		
	Spontaneously	4
	To speech	3
	To pain	2
	None	1
Best verbal response		
	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible words	2
	None	1
Best motor response		
	Obeys commands	6
	Localizes to pain	5
	Withdrawal	4
	Flexion to pain	3
	Extension to pain	2
	None	

FOUR Score

Table 3 describes the FOUR score.

Evaluation of Higher Mental Function

Speech and memory and dominant hemisphere: In intubated patients, evaluation of speech is difficult. Determining the dominant hemisphere in the acute scenario will be challenging. Possible history from relatives about handedness can give clues about the dominant hemisphere. Intubated patients can become irritable and restless due to inability to communicate. Allowing the patient to write or use medical charts to communicate can help in knowing and addressing the patient's discomfort. Also knowing the language patient is familiar with helps in evaluating the GCS and doing the neurological examination.

Sedation Interruption

The neurological examination is largely affected by the presence of sedation. If possible, interruption of sedation is advised followed by neurological evaluation. The sedation is reduced to an acceptable level in patients with raised intracranial pressure, agitation, and restlessness.

TABLE 3: FOUR score.

Eye response		
	Eyelids open or opened, tracking, blinking to command	4
	Eyelids open but not tracking	3
	Eyelids closed but open to loud voice	2
	Eyelids closed but open to pain	1
	Eyelids remain closed with pain	0
Motor response		
	Thumbs up, fist, or peace sign to command	4
	Localizes to pain	3
	Flexion response to pain	2
	Extensor posturing	1
	No response to pain or generalized myoclonus, status epilepticus	0
Brainstem reflexes		
	Presence of papillary and corneal reflexes	4
	One pupil wide and fixed	3
	Absent pupil or corneal reflexes	2
	Pupil and corneal reflexes absent	1
	Absent pupil, corneal and cough reflex	0
Respiration		
	Not intubated, regular breathing pattern	4
	Not intubated, Cheyne–Stokes breathing pattern	3
	Not intubated, irregular breathing pattern	2
	Breathes above ventilator rate	1
	Breathes at ventilator rate or apnea	0

Brainstem Reflexes

Brainstem assessment is vital in evaluating a patient. Brainstem reflexes include corneal, pupillary, gag, and cough reflex. These reflexes aid in assessing the functioning of the brainstem components. Other reflexes include oculocephalic, vestibulo-ocular, and menace reflex.

Examination of Cranial Nerves

Optic nerve examination:

- Fundoscopy: Papilledema indicates increased intracranial pressure.
- Visual fields: Determination of visual acuity in ICU patients will be difficult. The visual field cannot be accurately tested in patients with altered sensorium. Using Snellen's chart to see the visual acuity is not possible in the ICU.

Alternatives:

Menace reflex: It is a blink reflex to visual threat. In the patients who cannot participate in normal visual field testing, this reflex tests for normal visual processing.

It is elicited by a threatening hand gesture heading towards the eye while covering the contralateral eye to prevent its contribution to the menace reflex. A blink is considered a positive menace response.

Pupils: Pupillary size with its comparative size and reactions should be checked.

- *Widely dilated pupils (7–9 mm):* Midbrain lesions that affect the oculomotor complex or third nerve.
- *Medium size pupil (4–7 mm):* Primarily due to midbrain lesion or secondary to central herniation (e.g., tumor or hematoma in the supratentorial compartment)
- *Unilateral pupillary dilatation with deteriorating consciousness:* Sign of unilateral tentorial herniation
- *Small pupils (0.5–1 mm):* Pontine lesions
- Small pupils with loss of light reflex and upward gaze palsy—thalamic hemorrhage
- *Light reflex:* To see the pupillary constriction in response to light. To assess for direct and consensual light reflex.

Ocular position and movements: Examination includes—checking positon of the eyes, deviation from the midline, and to look for nystagmus. All the muscles of the eye are supplied by the third nerve, except for the superior oblique muscle and lateral rectus supplied by the trochlear and abducent nerve, respectively.

Trigeminal nerve:

- *Facial muscles and sensation:* Voluntary movements cannot be tested.
- *Trigeminal nerve:* It has three branches, two sensory ophthalmic, maxillary, and one motor, mandibular.
- Loss of function of the forehead and cheek due to ophthalmic and maxillary nerve lesions.
- Loss of jaw jerk is seen as a part of the lesion of the mandibular part of the trigeminal nerve.

Abducent nerve: The lesions involving the sixth nerve lead to lateral rectus palsy. The paralysis of the sixth nerve is considered a false localizing sign and can occur due to raised intracranial pressure due to any etiology. A possible mechanism for sixth nerve palsy is due to the long intracranial course and compression of the nerve at the petrous part of the temporal bone. Another possible mechanism proposed is a course of the sixth nerve direct from the brainstem as against the oblique or transverse course of the other nerves making this nerve more susceptible to injury.

Facial nerve:

- *Upper motor neuron (UMN) facial palsy:* Due to dual representation there will not be a loss of wrinkles on the forehead and the lower half of the face will be affected.
- *Lower motor neuron (LMN)-type facial palsy:* Flattening and loss of wrinkles on one side with a partial closure of the eyelid leading to uncovering of the sclera (lagophthalmos) is noted.
- *Bilateral diffuse cerebral lesions:* Primitive reflexes like sucking or snout reflexes are seen.
- *Corneal and conjunctival reflex*—absent bilaterally in a deep coma
- The afferent and efferent pathways for these reflexes include the trigeminal and facial nerves, respectively.

Auditory nerve:
Cochlear and vestibular component:

- *Cochlear component:* Damage to this component causes tinnitus or nerve deafness. Assessment by tuning fork method may not be possible.
- *Vestibular component:* Damage to this component can lead to vertigo and nystagmus. The vestibular function can be tested by using a cold caloric test to see the nystagmus. Nystagmus comprises two components: Fast and slow component. If cold water is irrigated, the eyes move toward the opposite ear. If the vestibular function is lost, the fast component is lost in case of brainstem dysfunction, resulting in the movement of the eyes toward the irrigated ear due to the slow component.

Glossopharyngeal nerve and vagus nerve: The nerve function can be tested by the gag reflex. The afferent for gag reflex is glossopharyngeal nerve and efferent is the vagus nerve. The absence of movement of the uvula in response to stimulation is indicative of an absent gag reflex. The lesions involving the brainstem affects gag reflex. In case of nerve dysfunction, uvula will deviate toward the normal side.

Spinal accessory nerve: Testing for nerve function is difficult in the intubated patient. The patient is told to shrug the shoulder and contraction of the trapezius muscle is observed with or without resistance. Testing of sternocleidomastoid is not possible.

Hypoglossal nerve: The hypoglossal nerve predominantly supplies muscles of the tongue. Nerve function can be tested by seeing the tongue movement or the presence of a deviation. Deviation of the tongue toward the affected side is seen in unilateral paralysis of the hypoglossal nerve.

Motor System

Tone: The tone is assessed during passive movement of the limb. Loss of tone is seen in the acute stage of the illness. Lesions of the pyramidal tract have hypertonia as against hypotonia in cerebellar lesions.

Conditions affecting muscle tone:

- *Neurogenic shock:* Neurogenic shock is characterized by loss of vasomotor tone below the level of injury. The clinical symptoms are characterized by vasodilatation, hypotension, and bradycardia.
- *Spinal shock:* This is seen in spinal cord injury. The clinical examination is characterized by loss of deep tendon reflexes and flaccid paralysis below the level of spinal injury with loss of bladder and bowel reflexes.
- *Cerebrovascular accident:* It is characterized by increased muscle tone of the affected side.
- *Disorders affecting neuromuscular disorders:* It is characterized by hypotonia or weakness.

The lesion affecting UMNs is characterized by increased muscle tone and hyperreflexia. The lesions affecting the LMNs cause weakness, loss of muscle tone, and areflexia.

Power: Muscle power is tested by seeing the muscle movements actively and after applying the resistance. The presence of proximal muscle weakness could be due to myopathies and distal muscle weakness could be due to neuropathies.

Muscle power is classified into different grades as described by the Medical Research Council (MRC) grading scales **(Table 4)**.

TABLE 4: Medical Research Council (MRC) grading.

Grade	*Response*
0	No contraction
1	Flickering movement
2	Active movement of the limb with the gravity eliminated
3	Active movement of the limb against the gravity
4	Active movement of the limb against the gravity and resistance
5	Active movement against full resistance

Reflexes

Testing of the reflexes is done in the normal way as it is an objective procedure. However, in patients with deep coma, the results may be interpreted differently.

In patients with deep coma:

- Absent deep tendon reflexes is noted on the paralyzed side.
- Superficial reflexes might not be elicitable and have little localizing value.
- Babinski's response—may be absent in patients with deep coma. However, unilateral Babinski's response is indicative of pyramidal tract lesion.

Sensory System

Detailed assessment of the sensory system is not feasible in intubated patients due to the inability of patients to communicate and cooperate with the procedure. With the limited assessment, loss of sensation or determining the level of sensory loss is possible.

Coordination

Assessment of gait disturbances or ataxia will not be possible.

Tests:

- Finger–nose test
- Heel to shin test
- Repeated movement

Meningeal Irritation

The presence of neck rigidity indicates meningeal irritation.

- *Kernig's sign:* Pain while doing a passive extension of the knee beyond 90°.
- *Brudzinski's sign:* Spontaneous flexion of hip and knee in response to flexion of the neck
- Meningeal irritation is seen in meningitis, cerebellar tonsillar herniation at the foramen magnum, or SAH.
- *Lhermitte's sign:* The electricity-like sensation radiating from the neck to the arms and leg is observed in multiple sclerosis.

Adjuvant to Examination

- *Computerized tomography (CT) brain:* This remains the initial modality of imaging in acutely altered mental status. It helps in identifying intracranial bleed.

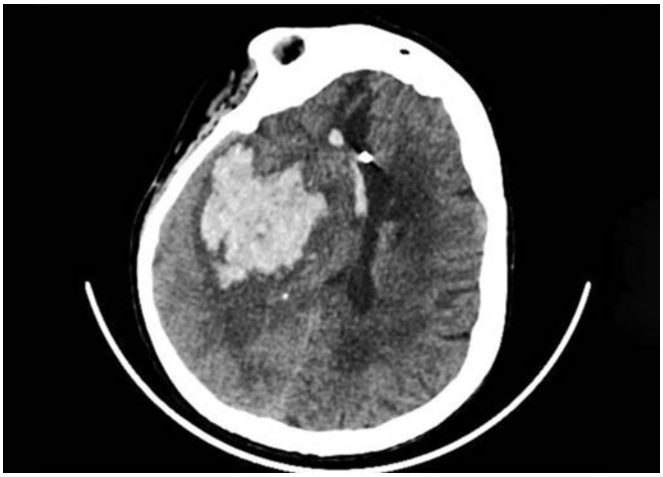


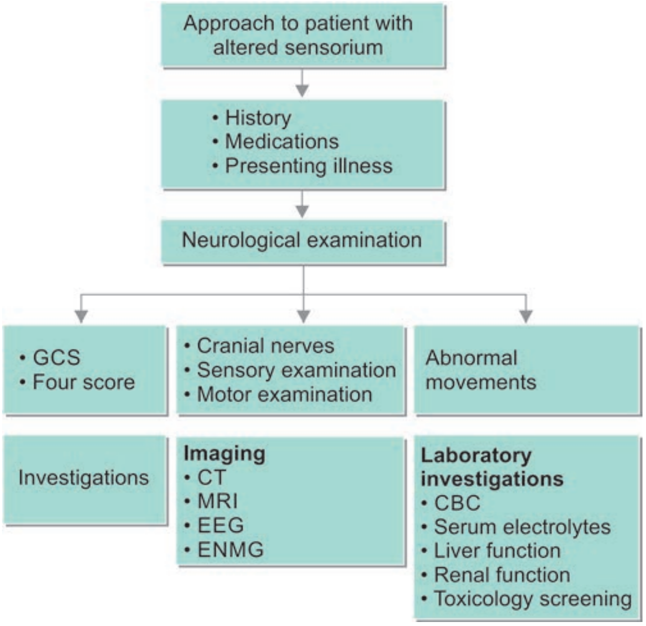
Fig. 2: CT brain with right gangliocapsular bleed.

If the CT brain is normal, doing a CT angio may give a clue about associated infarction, vasospasm, stenosis, dissection, or aneurysm. It can be used as an ancillary test in evaluation for the brain death. The absence of flow in the intracranial portion of the carotid arteries is pathognomonic of brain death.

In the patient described earlier, CT showed right gangliocapsular bleed with mass effect and midline shift **(Fig. 2)**.

- *Magnetic resonance imaging (MRI):* MRI helps in identifying acute infarction, lesions involving the posterior fossa or pituitary, osmotic demyelination, and fat embolism. MR venogram is the modality of choice for diagnosing CVT. MRI helps in identifying hypoxic-ischemic injury in postcardiac arrest patients. The newer modality such as a functional MRI is useful in identifying the dominant hemisphere, assessing speech function, and also in patients with epilepsy or space-occupying lesion (SOL) who are posted for surgery to know the vicinity of the lesion to the vital structures.
- *EEG (electroencephalogram):* Continuous or intermittent or as a screening test in a patient with altered sensorium to rule out nonconvulsive seizures

  Electroencephalogram helps in identifying ongoing seizures, titrating the antiepileptic medications, and is part of an ancillary test in brain death evaluation.
- *Lumbar puncture:* This is one of the initial investigations of choice in a patient presenting with altered sensorium. It helps in diagnosing various types of meningitis, autoimmune encephalitis, acute disseminated encephalomyelitis (ADEM), SAH, and malignancy.



Flowchart 1: Approach to neurological examination.

Approach to patient with altered sensorium
• History
• Medications
• Presenting illness
Neurological examination
• GCS
• Four score
• Cranial nerves
• Sensory examination
• Motor examination
Abnormal movements
Investigations
Imaging
• CT
• MRI
• EEG
• ENMG
Laboratory investigations
• CBC
• Serum electrolytes
• Liver function
• Renal function
• Toxicology screening

(CBC: complete blood count; CT: computed tomography; EEG: electroencephalogram; ENMG: electroneuromyography; GCS: Glasgow Coma Scale; MRI: magnetic resonance imaging)

- *Electroneuromyography (ENMG):* To identify neuromuscular illnesses such as Guillain-Barre Syndrome, Eaton–Lamberts syndrome, or ICU-acquired weakness (critical illness polyneuropathy). GBS with central nervous system (CNS) involvement is seen in Miller Fisher syndrome.
- *Transcranial Doppler (TCD):* This uses the Doppler principle to observe the velocity changes in the intracranial vessels. It is available at the bedside and can help to diagnose certain conditions like vasospasm and hyperemia. The diastolic reversal of the flow velocity is seen in brain death.
- *Optic nerve sheath diameter (ONSD):* This is a bedside test available for the diagnosis of raised intracranial pressure (ICP). The ONSD is measured 3 mm posteriorly to the globe. Various studies have described different cut-off values.
- *Digital subtraction angiography:* It is the gold standard for the evaluation of SAH.

Laboratory Investigations

Routine tests: Blood sugar level, complete blood count, serum electrolytes, and liver and renal function tests. Arterial blood gas test is done to identify metabolic or respiratory disturbances. Toxicology screening is also done as applicable.

CONCLUSION

- Neurological examination of ICU patients comprises detailed history taking with emphasis on chronology.
- Focused clinical examination aids in localizing the site of the lesion.
- Additional evaluation by laboratory testing and imaging modalities supports the clinical findings and helps in identifying the etiology and plan further management. Algorithmic approach can help in ensuring complete neurological evaluation **(Flowchart 1)**.

ACKNOWLEDGMENT

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Chapter

17

How will I Examine a Patient with Polytrauma?

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ABSTRACT

History, physical examination and stabilization of vitals in polytrauma patients go hands on hand. History taking in trauma patient mostly rely on bystanders, relatives or from patient (preserved mentation). AMPLE and MIST are the mnemonics used to cover basic history taking and to understand mechanism of injury. After stabilization of vital parameters (identification and correction: part of primary survey in trauma), it is important to cover head-to-toe examination (part of secondary survey in trauma). Positive physical examination should be evaluated further by relevant imaging techniques like ultrasonography, CT scan or MRI.

As William Osler said "*listen to your patient*, he is telling you the diagnoses", if we extend this statement, not only listen but observe your patient too to reach the conclusion. This emphasizes the value of history and examination in medical practice.

INTRODUCTION

Examination of polytrauma patient is an art that evolves with practice. The difference is that occasionally trauma patient are unable to give history or valid presentation of symptoms. It is also observed that history is given by bystanders about event of injury and timeline journey. It is pivotal to stabilize the patient simultaneously with examination ongoing. And if needed, examination should be repeated based on need (like to identify source of bleeding/hemorrhage), development of new symptoms, and change in vital parameters. In this chapter, we will discuss relevant history questions, precautions to consider before examinations, general examinations, and systemic examinations of polytrauma patients. We discuss in the format of what to see, why to see, and how to see for that relevant question whenever it is applied.

CASE SCENARIO

A 27-year-old male was brought to the emergency room after a fall from the terrace while kite flying. He lost consciousness at the site of the accident observed by his father. On arrival at the emergency (half an hour of the incident), the patient vomited twice mostly of food particles. He opened his eyes on call, verbalized his name and followed commands by lifting both arms but prefer to sleep. He has pain and tenderness in the back side of neck. He has bruise and swelling on his left wrist joint, left thigh and ankle. A quick primary survey did not reveal any problem with ABC (airway, breathing, and circulation).

History and Symptomatology

History in polytrauma cases is different than other intensive care unit (ICU) cases, sometimes we are getting history from bystanders or by relatives (here father) after a couple of hours on the arrival of patient.

What to ask? AMPLE history is a part of the secondary survey, which includes: *A*llergies, *M*edications currently used, *P*ast illnesses/*P*regnancy, *L*ast meal and *E*vents/*E*nvironment related to the injury.

Why? Allergic history will help to avoid particular drugs (sedation, antibiotic, and paralytic agent) or food on subsequent days of critical care. Last meal intake decides the method of intubation and readiness for risk of aspiration. If uncertain about the last meal, in such cases better to choose rapid sequence intubation (if airway is compromised or decision of intubation made). History of comorbid conditions and medications gives idea about reservoir of system, prognostication, and part to optimize medical management. Certain medications need to be continued like thyroid medications. Sugar management should be switched from oral [oral hypoglycemia agent (OHA)] to intravenous/subcutaneous (IV/SC) insulin if patient kept NBM (nil by mouth)/changing dynamics of ICU. Environment history is particularly very important in case of fire injury and war casualties. Closed area burn is prone for carbon monoxide poisoning. Time to reach to hospital and what primary aid/treatment patient received also play crucial role in further management.

What to ask? MIST is another acronym that includes: *M*echanism (and time) of injury (in my patient fall from height), *I*njuries found and suspected, *S*ymptoms–Signs and *T*reatment initiated.

Why? Mechanism of injury like high-speed motor vehicle injury/collision associated with more severe injury and cervical spine involvement. Fall from height associated with spine, calcaneal, and/or pelvic injury (more relevant for our case: Fall from height). **Table 1** shows mechanism of injury and associated/likely part/organ of body involvements.

What are the symptoms and their relevant clinical significance? Headache, visual blurring or diplopia, fluid running from nose or ear, dizziness, imbalance while walk, vomiting, or nauseating sensation are the symptoms of head injury [likely high intracranial pressure (ICP)] (our patient had vomiting and event of unconsciousness). In case of difficulty in breathing or pain while expanding chest, it is important to focus on airway and thoracic injury, i.e., tracheal injury, pneumothorax, and rib fracture. Chest heaviness or angina-like manifestation is seen in case of blunt chest injury or in case of cardiac tamponade. Pain and swelling in specific area with tenderness is suggestive of underlying soft tissue damage or bone fracture, which requires further evaluation and imaging. Painful neck movement/tenderness on spine area should raise suspicious of spinal injury. Inability to move lower limbs or abnormal sensory perception gives clue about neuronal injury and required evaluation. Pale and swollen limb with abnormal/painful sensation should raise the suspicion of compartment syndrome.

TABLE 1: Mechanism of injury.

Fall from height	• Axial spine injury • Calcaneal injury/bilateral lower limb injury • Pelvic injury
Front side injury/motor vehicle injury	• *Steering injury/dashboard injury:* Flail chest, traumatic aortic injury, pneumothorax, and myocardial contusion • *Windscreen injury:* Facial injury and head injury
Side injury/ motor vehicle injury	Diaphragmatic injury, abdominal visceral injury, and pelvic and lower limb fractures
Rear injury/ motor vehicle injury	• Head injury • Cervical spine injury
Pedestrian injury by vehicle	All possible injuries likely
Penetrating injury/gunshot injury	• Depends on area involved • *Thoracic:* Hemothorax and pneumothorax • *Abdomen:* Solid organ injury like splenic, liver, or hollow viscera injury

Source: Modified from Student Course Manual ATLS®. Advanced Trauma Life Support®. American College of Surgeons; 2018.

Before General Examination

What to do? Wear protection devices: Why? Many polytrauma patients had exposure of chemical substances or irritable substances/poisoning materials. It is noteworthy to wear gloves and keep personal protection devices (COVID era) ready before the examination any of polytrauma patients. Hand hygiene and aseptic handling of open wound should be maintained.

What to do? The presence of female coworker or nursing staff is must for female patient examination and the privacy of patient must also be kept in mind during examination.

What to ask? Clinicians can quickly assess A, B, C, and D in a trauma patient (10-second assessment) by identifying themselves, asking the patient for his or her name, and asking what happened. Observe whether the patient having any focal deficit in terms of left/right side of limb weakness.

Why? Giving the right answer about describing himself verbally and recalling memories of event shows airway patency, good cerebral perfusion, and mentation. Talking without pause/completing sentences smoothly shows good respiratory reserve.

Checking Vitals

Stabilization of vital signs is always target of primary survey in trauma. Vital sign response to resuscitation helps to categorize responder, transient responder, and nonresponder.

- What? Monitor heart rate and blood pressure: Why? A rapid, thready pulse is classically a sign of hypovolemia.

Heart rate and blood pressure helps to categorize/understand severity of hypovolemic shock. It should be kept in mind that beta-blockers and hypothyroidism-like condition are the confounding factors on assessing heart rate response to hypotension/hypovolemia. Wide pulse pressure is a sign of distributive shock while narrow pulse pressure is seen in hypovolemic shock/cardiac tamponade.

- What? Monitor respiratory rate and oxygen saturation: Why? Pulse oximetry gives idea about patient oxygen saturation and gauge crude idea about perfusion. Hypoxemia should be evaluated and treated simultaneously with oxygen devices. It should be kept in mind pulse oximetry does not give any idea about ventilation or hypercarbia. Monitoring respiratory rate and pattern in patient of polytrauma give insight about work of breathing. High respiratory rate can be seen due to pain, lung pathology, or as part of respiratory compensation (in view of oxygen demand supply).
- What? Monitor temperature: Why? Hypothermia is seen quite often in polytrauma cases. Exposure to environment, large volume resuscitation with saline/blood products leads to hypothermia. In such cases gradual rewarming of patient and using warmer device for fluids aid in mitigation of one of the components of lethal triad.
- What? Monitor urine output: Why? (If Foley catheter present) Adequate urine output >1 mL/kg/h is suggestive of good urinary perfusion. Change of urine color (red or amber/brown) is seen in condition like acute tubular necrosis (due to hypotension-related acute kidney injury) or in cases of rhabdomyolysis/compartment syndrome/myoglobinuria.
- What? Glasgow coma scale (GCS): Why? GCS helps to categorize traumatic brain injury. It also helps in the decision of invasive ventilation (if GCS 8 or less). It consists of three parameters: Eye-opening response, motor movement response, and verbal response. It is important to rule out drug effect (sedation/analgesia/paralytic agent/poisoning) to get reliable GCS score.

General Physical Examination

Head-to-toe Examination Also Part of Secondary Survey

Gross examination/general appearance/skin:

What? Checking skin color and temperature is a very important aspect to check perfusion in trauma patient.

TABLE 2: Injury size and type and antibiotic choice.

Wound <1 cm; minimal tissue damage/contamination	First generation of cephalosporins
Wound 1–10 cm; moderate tissue damage; communication of fracture	Add clindamycin or in patient of penicillin allergy
Severe soft tissue damage and contamination *plus* associated vascular injury	Add aminoglycosides
Farmyard, soil, or standing water contamination (irrespective to size or severity)	Broad-spectrum antibiotics Piperacillin + tazobactam

Why? Cold and calmy extremities with pale color of limbs are suggestive of hypoperfusion of tissue and need evaluation and resuscitation. Cyanosis/bluish discoloration of face/lip or finger tips is suggestive of hypoxemia and should be immediately address with oxygen delivery device. Capillary refill time is a good bedside tool that gives idea about perfusion status of patient and helps in resuscitation strategies.

What? Type and size of wound

Why? Type and size of wound give idea about degree of injury, antibiotic management, and surgical plan. Contaminated wound or open wound needs tetanus prophylaxis. Size of wound and contamination material decide type of antibiotic. **Table 2** shows injury size and type and antibiotic choice according to guideline.

What? Presence of blood at urethral tip or bruise on pelvic area

Why? It is suggestive of urogenital injury, in such cases catheterization of urethra should be avoided to prevent injury/false track.

What? Visible bony deformity or visible fractured bone with blood exsanguination

Why? Such area of active blood exsanguination if identified on general examination, they should be pressed with roller gauze/pressure tamponade to be applied for source control.

SYSTEMIC EXAMINATION

Examination of Head

What to examine in head?

- Facial deviation, ptosis, pupillary size/asymmetry, speech, or vision abnormality should be checked.

Why to examine?

- A focal deficit may require neuroimaging like computed tomography (CT)/magnetic resonance imaging (MRI) brain. Visible depressed fracture of skull is associated with underlying brain parenchymal damage/contusion.

What to examine in pupil? Reactivity of pupil to light and symmetry/size of pupil are the basic steps for neurological examinations.

Why to examine? Asymmetry and sluggish/nonreactivity to light response are considered poor sign and should be managed immediately with measures to reduce ICP.

Raccoon eye: Unilateral or bilateral progressive periorbital ecchymosis with edema is known as the "raccoon eyes" and the "panda sign". It is commonly associated with fractures of the base of the anterior cranial fossa.

Nose bleeding/rhinorrhea: Cribriform plate fracture should be suspected in such cases; anterior packing of nasal cavity should be done. It is noteworthy that nasogastric tube should be avoided in such cases of nasal/suspected basal skull injury.

Distorted anatomy of mandible or maxilla/facial bones: Many polytrauma presented with facial deformity, lacerated wounds, and dislocated jaw. In such cases expect difficult intubation and keep difficult airway cart standby.

Examination of Neck

- *Inspection:* Patients with maxillofacial or head trauma should be assessed for cervical spine injury too. If patient unable to move neck side by side or having painful movement, it is must to rule out cervical spine injury. Painful neck movement needs restriction of neck movement with collar. Cervical X-ray or CT cervical spine is needed to rule out spine injury. Do not attempt doll's-eye testing until a cervical spine injury has been ruled out. Head tilt-chin lift maneuver also avoided in suspected case of cervical spine injury. Facial deformity, mandible or maxillary bone fracture make intubation difficult. Tracheal shift, carotid pulse, jugular venous pressure, and subcutaneous emphysema should be checked while neck inspection. Increased jugular venous pulse is suggestive of cardiac tamponade/tension pneumothorax while collapsed jugular venous pulse is associated with hemorrhagic shock/massive hemothorax. If patient is conscious and following verbal command, it is important to observe oral cavity and opening. Oral cavity opening will decide about Mallampati grading to predict ease of intubation. In our case, patient had painful neck movement with tenderness, need evaluation by doing imaging cervical X-ray or CT-scan.
- *Palpation:* Unresponsive to verbal or tactile stimuli patient should be checked for carotid pulse. Absence of central pulse needs urgent cardiopulmonary resuscitation. Deviation of trachea is normally seen in pneumothorax, large hemothorax, and lung collapse. This can be checked by pressing fingertips between lateral tracheal wall and medial portion of sternocleidomastoid muscle, comparing one side with other. Subcutaneous emphysema on palpation is suggestive of airway injury or coexistence of pneumothorax.
- *Auscultation:* In case of foreign body in airway or obstruction in trachea/pharyngeal area, give rise to audible stridor. Such cases need evaluation of oral cavity or pharynx. Gurgles is suggestive of collection/retention/pooling of secretion in airway/pharynx, requires suctioning and airway protection. Hoarseness in voice is suggestive of functional laryngeal obstruction.

Examination of Thorax

- *Inspection:* Abnormal breathing pattern like abdominothoracic breathing, accessory muscle utilization, and paradoxical breathing should be checked in polytrauma patient having respiratory failure. Bruise/blunt trauma on chest or back area is likely associated with underlying tissue damage. Abnormal chest rise/decrease/unequal chest rise gives idea about lung collapse/single lung ventilation or pneumothorax. Depressed ribs are likely associated with internal organ damage, i.e., lung contusion, liver, or splenic injury.
- *Palpation:* Palpation of apical pulse gives idea about heart position that can change in case of pneumothorax or hemothorax/mediastinal shift. Palpation is also important in the assessment of ventilation. With palpation symmetry and volume of breath can be assessed. How? This can be done by placing thumbs together at the midline and observing movement while breathing cycle. Normally thumbs separated by 2–3 cm.
- *Auscultation:* Auscultating lung area gives idea about bilateral equal air entry. That can be absent/decrease

in pathology like lung collapse or large hemothorax. Muffled heart sound can be heard in pericardial effusion or cardiac tamponade. Crepitation/Crackle can be heard in case of pulmonary edema while wheezing can be heard in case of bronchospasm or reactive airway.

- *Percussion:* The lung is filled with air so percussion of it gives a resonance sound. Hyperresonant note seen in case of air trapping like in emphysema or in case of spontaneous pneumothorax. Dull note is suggestive of absence of air seen in atelectasis or pleural effusion.

Examination of Abdominal Area

- *Inspection:* Distended abdomen is seen in hemoperitoneum or paralytic ileus. Post bag and mask ventilation also give rise to distended abdomen (aerophagia). Periumbilical bruising (Cullen's sign) is seen in hemoperitoneum. Bruise on abdominal area should raise the suspicion of intra-abdominal organ injury. Pelvic area is always a neglected entity in examination. Bleeding per urethra is suggestive of urogenital injury. Ecchymotic area over iliac wings, pubis, labia, or scrotum should point toward pelvic fractures and should be evaluated.
- *Palpation:* Tenderness and involuntary rebound rigidity on palpation are suggestive of irritation likely seen in perforation/peritonitis. Crepitus on palpation is suggestive of air in subcutaneous tissue.
- *Percussion:* Percussion over liver and spleen area will give idea about size/enlargement of organ area. Increased dull note should raise alarm about hematoma of solid organ.
- *Auscultation:* Absent bowel sound seen in paralytic ileus while hyperactive bowel movement (borborygmi) is usually associated with small bowel obstruction.

Examination of Limb/Extremities

- *Inspection:* Restricted movement distal to joint, swelling, and painful movement should be evaluated for fracture. Discoloration of skin black/blue is suggestive of decreased perfusion to tissue. Contaminated wound should be thoroughly washed and antibiotic is needed.
- *Palpation:* Major distal vessels should be assessed for pulse. In case of absence, immediately surgeon or interventional specialist should be involved. Compartment syndrome gives rise to decrease/absent pulse volume. Capillary perfusion checks also give idea about perfusion and resuscitation target. Tenderness and swelling on joint or bony area should require X-ray for bony injury/fracture.

In our case, patient had bruise and swelling over left thigh, wrist, and ankle. It is advisable to go for X-ray of relevant area to rule out fracture. Femur fracture is frequently associated with vascular injury/massive blood loss. Checking distal pulse of limb and color changes is also crucial in our patient in view of thigh and ankle injuries.

POLYTRAUMA PATIENT IMAGING BASED ON HISTORY AND EXAMINATIONS

Any polytrauma patient with moderate to severe traumatic brain injury should undergo CT brain. In our patient presence of loss of consciousness and vomiting mandate need of CT scan brain. "Trauma triple imaging" like cervical spine X-ray, chest X-ray, and pelvic examination with both hips should be done in polytrauma patients. Fall from height (>3 feet) should be evaluated with cervical X-ray, pelvic X-ray, and calcaneal (ankle) X-ray (applied to our patient). Extended focused assessment with sonography in trauma (eFAST) includes lung ultrasound with four major area: Cardiac view, pelvic view, right, and left upper quadrant of abdomen. In absence of ultrasound and hemodynamic instability, diagnostic peritoneal lavage should be done to detect hemoperitoneum. Positive FAST with stable hemodynamic patient should undergo CT abdomen with pelvic with IV (intravenous) contrast.

SUMMARY

- Take necessary precaution and permission before starting physical examination of patient.
- In polytrauma case examination: History, examination, and patient stabilization go hand on hand.
- It is always advisable to repeat examination when in doubt or clinical diagnosis is not met.
- Relevant imaging should be asked whenever any positive findings for confirmation of diagnosis and fasten the intervention.

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Chapter

18

How will I Examine a Patient after a Complex Surgery?

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ABSTRACT

Patients undergoing complex surgeries often have physiological derangements needing multidisciplinary intensive care unit (ICU) care. Appropriate history of patients' preoperative status and intraoperative events is important for a better understanding of the patient's further course. A quick, focused, and systematic examination of these patients helps in the identification of problems and facilitates treatment. The physician should follow a systematic approach to examine critically ill patients after surgery. This chapter will focus on what to examine in critically ill patients' postsurgery including noting down specific history, signs, and symptoms and systematic clinical examination of the patient in the ICU.

INTRODUCTION

Patients requiring intensive care after surgery are a frequent occurrence in any intensive care unit (ICU). The patients after surgery are admitted to ICU either because of the patient risk profile, the complexity of the surgery, or both. Also, there are circumstances where the patients admitted to ICU may need emergency surgery. Getting relevant history, a quick focused examination that will help diagnose and manage, is very important.

Most of the time the patients who need ICU care will have systemic inflammatory response syndrome (SIRS), fluid shifts, hemodynamic derangements [e.g., Whipple surgery, retroperitoneal lymph node dissection (RPLND), liver resection etc.], need organ support (organ failures) [e.g., right ventricular (RV) failure following pneumonectomy, liver failure following liver resection/complex intraabdominal surgery in patients with compromised liver function, renal failure, vasoplegia, etc.], multiorgan dysfunction with critical illness to begin with. So, it is very important for the ICU physician to know about the patient risk factors, risk stratification, and anticipated complications in the perioperative period. Also, it is equally important to know about the steps of surgical procedures, intraoperative events, and anticipated postoperative needs. The examination of patients after surgery can be preplanned, e.g., during routine bedside rounds in ICU or unplanned on demand/during urgent situations. Considering the complexity of the situation and the urgency of treatment needs, the examination should be focused, rapid, systematic, and individualized for specific surgical procedures.

CASE SCENARIOS

1. *A 45-year-old gentleman had a history of abdominal pain and vomiting for 5 days. He was treated in a nearby hospital for 8 days for gallstone-related acute pancreatitis. He underwent endoscopic retrograde cholangiopancreatography (ERCP) and the removal of the common bile duct (CBD) stone. He required a short ICU stay and was discharged home on day 10 of his illness. After a week at home, he came to the emergency room (ER) with severe abdominal pain, nausea, and breathing difficulty for 3 days. In ER, on examination, he had a heart rate of 142 beats/min, blood pressure of 94/70 mm Hg, and respiratory rate of 28 breaths/min. He had pH of 7.252, partial pressure of arterial carbon dioxide ($PaCO_2$) of 48 mm Hg, bicarbonate of 18 mmol/L, and lactate of 5.2 mmol/L. Contrast-enhanced computed tomography (CECT) abdomen revealed acute necrotizing pancreatitis, gross collection in the left paracolic gutter, lesser sac, and diagnostic tap showed purulent aspirate.*

Hypothetical course A	*Hypothetical course B*
The patient was taken for surgery. He underwent necrosectomy and drainage of purulent collection *Intraoperative events:* (A) Worsening shock, low urine output, and hypothermia. He was shifted to ICU on ventilator, noradrenaline 0.8 µg/kg/min and vasopressin 1.8 U/mL/h	The patient was taken for surgery. He underwent necrosectomy and drainage of purulent collection *Intraoperative events:* (B) Apart from described in (A), he had persistent oozing from the operated site despite the best possible hemostasis. The bowel loops were distended but healthy. The abdomen was left open with pressure paddings placed for scheduled relook after 48 hours

2. A 62-year-old had a habit of smoking for 30 years, a known case of diabetes, hypertension for 12 years, ischemic stroke 3 years back, no residual weakness, and coronary artery disease for 2 years and had a myocardial infarction 8 months before. He is having dilated left ventricle, left ventricular ejection fraction of 45%, and limited activities of daily living. He is a known case of carcinoma esophagus, postchemotherapy. He was admitted to ICU after transthoracic esophagectomy. Intraoperatively he had hypoxia after positioning, atrial fibrillation and hypotension requiring vasopressors. He was shifted to ICU admission on a controlled mode of ventilation and noradrenaline support of 0.4 *µg/kg/min*.

History and Symptomatology

History should start with the details of the present surgery, which includes the intraoperative details of events, current detail of physiological derangements, organ supports, and preoperative patient-related factors [activities of daily living, organ dysfunctions, METs (metabolic equivalents), previous surgical history, ICU admission details, etc.]. *See* **Tables 1 and 2** for details.

General Physical Examination

The general examination should be rapid and specific. Look at the vitals from the multipara monitor and intervene if there are any cardiorespiratory compromises. Rapid head-to-toe examination, assessing for pallor, cyanosis, and jaundice followed by looking for signs of the adequacy of global perfusion, signs of bleeding (oral cavity, surgical side, and drains), temperature (hypothermia), bed space, monitoring devices, and infusion pumps (analgesia, sedation, insulin, and any other). *See* **Table 3** for details.

Systemic Examination

There is no specific sequence to follow, but we must examine all the systems. It is logical to begin with the system where the surgery is performed/planned. It is equally important to assess the nutritional status and pain, both should be an extension to systemic examination in critically ill surgical patients. It is accepted practice and bedside teaching to follow a certain sequence of examination, i.e., inspection (see), palpation, percussion (feel), and auscultation (listen).

Inspection

Look at the surgical site, suture lines, and the nearby area in sequence.

- *Suture line:* Look for intactness of sutures, suture technique—tight sutures, reinforced, tension sutures, open skin, Bogota bag, etc. **(Figs. 1 to 3)**. These will give an idea of the difficulty in approximating the skin, intravisceral pathology, and anticipated complications.
- *Skin color:* Redness or discolorations may indicate infection/inflammation, ecchymosis, or hematoma. This may give a clue about the underlying collection in deep layers. In ventilated patients, localized skin discoloration may be the only sign of underlying pathology. Maceration of surrounding skin or erosion often indicates prolonged exposure to moisture or proteolytic enzymes. It is usually white but red when inflamed.
- *Exudate/discharge:* Exudate is normal and often present in normal healing would. When it is excessive or abnormal it is of concern. It is commonly seen in ulcerative wounds, burns, and postdebridement for cellulitis/necrotizing fasciitis. The discharges from the operated site are either serous, purulent, blood or combination. Careful follow-up will help to find the pathology at the earliest.
- *Distension:* Any swelling or local distension near the suture line—a collection of blood, fluids, or pus. Gaseous distension and expanding contents (fluid, gas, and tissue) will give a clue of underlying pathology.

TABLE 1: Relevant history and symptomatology in critically ill patients after surgery.

Domain	***Relevant history to note***	***Comments***
Present surgery	*What surgery:* • Open/laparoscopic *Intraoperative events:* • *Anesthesia and patient-related:* – Difficult airway – Body habitus – Organ dysfunction and support – Preoperative functional status – Hypotension and hypoxia – Fluids, medications, I/O, and blood products • *Surgery-related:* – Steps of surgery and related events – Injuries and repairs – Drains and their positions – Specific instruction for surgical teams	Intraoperative events like hypotension, vasopressor needs, endotracheal tube adjustment, bronchospasm, intraoperative ECG, echo findings, vascular injuries, blood product consumption, site of anastomosis, etc. are important to note to know about the patient's status and plan further management
Preoperative status	*Baseline functional status:* • Activities of daily living • METs • Nutrition status *Comorbidities:* • Are comorbidities under control? – Optimized with medications - For example, in scenario 2, it is important to check whether the cardiac function was optimized with medications or still needs further titration of medicines, e.g., ACEIs/beta blockers to achieve target blood pressure and heart rate - Antiplatelet for stroke – Whether the comorbid conditions warrant further evaluation to know the optimal status? - *Scenario 2:* Any cardiology plans for a coronary angiogram or treadmill test to look for critical stenosis? Not because the patient is going for surgery but as part of the standard management protocol *Organ dysfunction/support:* – Whether the patient is mechanically ventilated, on dialysis or vasopressors in the immediate preoperative period (common in patients from ICU) – Abnormal laboratories	• The preoperative functional status influences the patient's outcome and recovery after a severe illness and after any surgery. These will help in risk stratification of cardiac patients going for noncardiac surgery, pulmonary functions tests, FVC, 6-MWT to help predict postoperative pulmonary complications. *(Scenario 2)*. Preoperative nutrition status will guide nutrition plans postoperatively, especially in case of emergency surgeries • Postoperative complications can be less in well-optimized patients for their comorbidities. It may not be possible to get fully optimized patients, especially in semiemergency settings like oncological surgeries where time is important to prevent the advancement of the disease
Current physiological derangements	• Hypoxia, hypotension—cause, what actions were taken. *(As in scenario 1)* • Blood loss—current Hb, platelets coagulation profiles *(As in scenario 1 B)* to enquire steps to control oozing) • Intraoperative cardiac arrest, ACS, heart blocks, etc.	ICU physicians should know about the physiological derangement that may help titration of medications and follow-up
Organ supports	*How much organ support?* • Oxygen needs, vasopressor needs, etc. • Blood product's needs, Hb, and platelets coagulation profiles • On pacemaker/cardiac output monitors, internal defibrillators, ECMO, etc.	Many times, patients will be ventilated and will be on vasopressors. Certain occasions may need urgent dialysis and correction of coagulopathy

Contd…

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Domain	***Relevant history to note***	***Comments***
Multidisciplinary communications	• Need for the continuous drain of NG, imaging at predefined time, and nutrition plans • Analgesia plan, the timing for removal of epidural catheter, drugs that are given during transport, antibiotic timings, etc. • Cross-references that were given to other disciplines. • Specific follow-up instructions	These are very important to plan the treatment of the patients. It is better to document this communication, these are most often missed!

(ACEIs: angiotensin-converting enzyme inhibitors; ACS: acute coronary syndrome; ECG: electrocardiography; ECHO: echocardiography; ECMO: extracorporeal membrane oxygenation; FVC: forced vital capacity; ICU: intensive care unit; I/O: input-output; METs: metabolic equivalents; NG: nasogastric; 6MWT: six-minute walk test)

TABLE 2: Signs and symptoms to be elicited in critically ill patients after complex surgery.

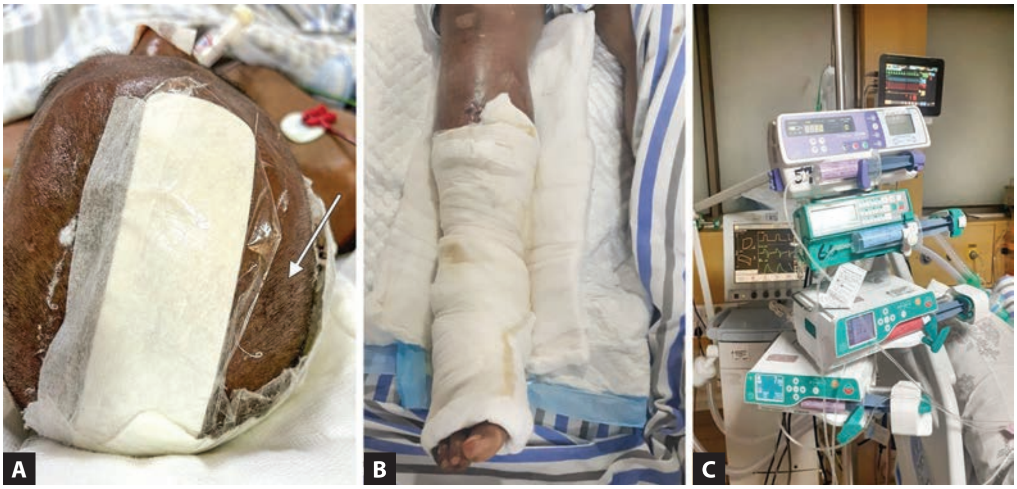
Domain	***Eliciting of signs/symptoms***	***Comments***
Pain	• Pain is the most common symptom expressed by any painter after surgery • A verbal/visual analogue scale is commonly used • In critically ill patients on mechanical ventilators, it is a real challenge in assessing the pain. Common scales used are CPOT/BPS	Analgesia (opioid) is always provided to all patients, but often it is not titrated to individual patient needs. Assessment of pain should be an active process
GI	• Nausea/vomiting: Visualize the color of blood/bile • Altered bowel habits • Constipation or reduced bowel movement is common • Abdominal distension (paralytic ileus)	Patients after complex surgeries or admitted to ICU requiring emergency surgery may have GI symptoms irrespective of the site of surgery
Cardiorespiratory	• Paradoxical breathing/breathing difficulty—due to splinting of (chest/abdomen—respective site of surgery) • Arrhythmias—thoracic surgery *(scenario 2)*	• Adequate pain relief is mandatory to avoid basal atelectasis in thoracic-abdominal surgeries • Watch for breathing patterns (paradoxical breathing, splinting of ribcage/abdominal wall)
Central nervous system	Screen for postoperative cognitive dysfunction and delirium (CAM-ICU is frequently used)	It is common to develop delirium in the postoperative period and this will be more when critically ill
Musculoskeletal	Signs of muscle damage—rhabdomyolysis post-trauma/reperfusion injury Inspect the limbs with traction, ribs, and spine post-trauma	Surgery in trauma patients is challenging due to multisystem involvement. All injuries and management should be kept in mind Vascular surgeries are at risk of reperfusion injury
Limbs and vascular	• Sins of hypoperfusion—cold peripheries, prolonged CRT, mottling, etc. • Swollen legs—DVT/immobile/dependent • Pain and discoloration—orthopedic and vascular surgery	• All patients are at risk of DVT, especially after surgery. So specific signs and symptoms should be assessed daily • The peripheral pulse should be felt in case of limb/orthopedic surgery, especially after trauma fixation

(BPS: Behavioral Pain Scale; CAM: confusion assessment method; CPOT: Critical Care Pain Observation Tool; CRT: capillary refill time; DVT: deep venous thrombosis; GI: gastrointestinal; ICU: intensive care unit)

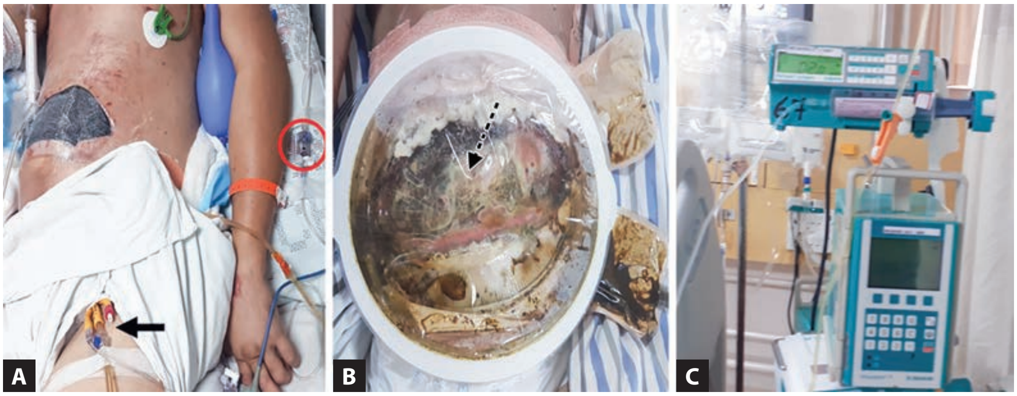
TABLE 3: General examination of patient postsurgery.

Domain	***What to examine***	***Comments***
Head and neck	• *Facies:* Always look at the patient's face, it may give many clues – An anxious and unhappy face will tell you something is not going well! – Sunken eyeballs, pale and dull face → severe dehydration and shock – Anxious, restless, agitation—delirium • Eyelids and orifices—ENT bleeding—in head trauma • Dilated neck veins—increased intrathoracic pressure • Oral cavity—do not forget to check for oral hygiene, ulcers, bleeding, and candidiasis • Feeding tubes, endotracheal tube, and necklines—fixation, the color of fluids if any • Pallor, icterus, cyanosis, and lymphadenopathy • Nutrition—buccal fat, temporalis muscle, and the oral cavity for signs of malnutrition	• The general examination should start with vitals from the bedside monitor. In critically ill patients, the normal vitals in monitors will reassure them that things are going well, and no urgent intervention is needed • The examination can be focused—individualized based on the patient. (Neurosurgery—head-end elevation and EVD positions, cardiorespiratory—trachea position, necklines, etc.)
Extremities	• Perfusion signs • Vascular access • Peripheral edema • Injuries • Slabs/casts—orthopedic surgeries • TEDS and SCDs	• Patients' extremities give a clue of the adequacy of perfusion; look for the tip of digits for cyanosis, and embolism • Check for swelling—pitting edema (third spacing, fluid overload, severe capillary leak, and SIRS)
Thorax and abdomen	• Respiration • Dressings • Back and Perineal area—Foley's catheter, pressure sores	• *Breathing pattern:* It is important to look for any paradoxical or uneven breathing pattern. It will be the first step for further detailed examination • Often missed areas are the genitals and perineum—penile skin retraction, pressure sores, and abscesses are commonly missed
Temperature	Core temperature may be vital in case of prolonged surgery to rule out hypothermia and postcardiac arrest to maintain temperatures in the target range	• Temperature should be monitored in all surgical patients and charting should be done • Warming devices should be checked for appropriate functioning
Bedspace	• Infusion pumps—insulin, analgesia, sedation, vasopressors, epidural/PCA, etc. • Monitoring devices • Drainage bags	• We should not forget to look at the bedspace • Many times patient will be on titrating infusions—heparin infusion postvascular surgeries, pressure monitoring devices, EVD, etc.
Posture	Look at the posture of the patient • The patient may adapt certain postures, e.g., may bend forward, or roll to side as a part of adaptation to the pain of pancreatitis, renal colic, etc. • Patients on mechanical ventilation may have flexion of legs, and upper extremities—which may indicate pain (BPS) • Patient may need certain posture to maintain as part of treatment strategy—burns patient with a skin graft, orthopedic patients, sacral pressure sore debridement surgery, etc.	• This may give a gross idea about patient status (pain) • Frequent positioning may increase the risk of tube/lines/drain displacements

(BPS: Behavioral Pain Scale; ENT: ear, nose, and throat; EVD: external ventricular drain; PCA: patient-controlled analgesia; SCD: sequential compression devices; SIRS: systemic inflammatory response syndrome; TEDS: thromboembolic deterrent stockings)

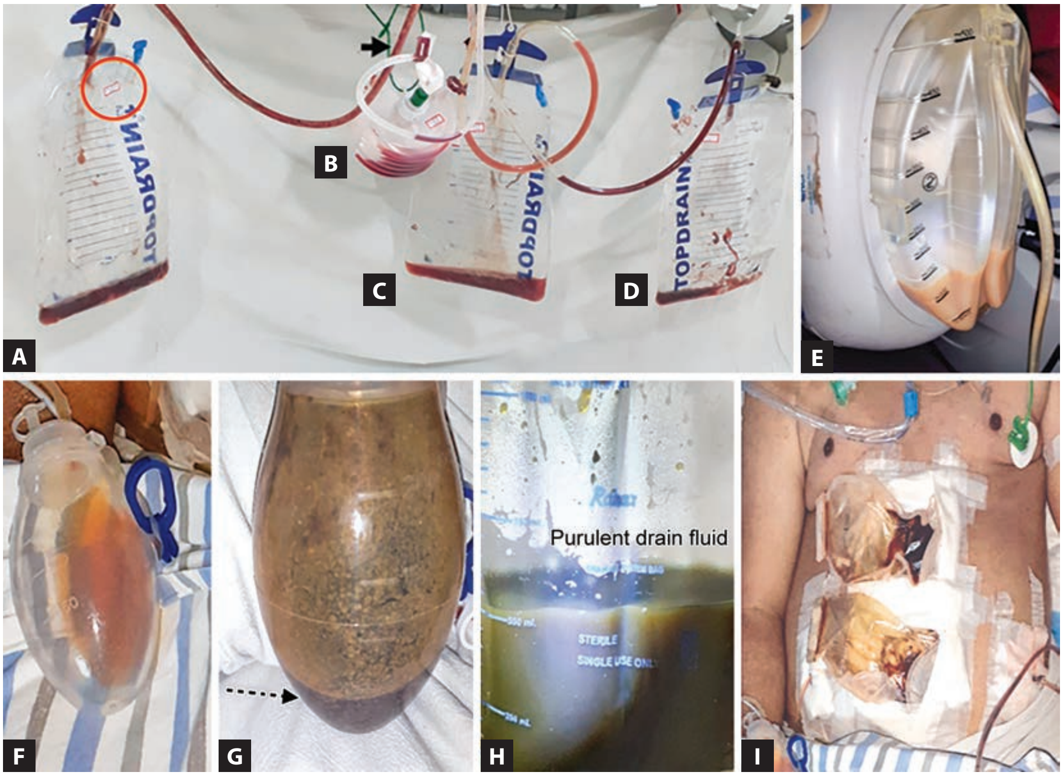


Figs. 1A to C: The postoperative site with bedspace. (A) A patient with traumatic brain injury postcraniotomy and decompression craniectomy in the late postoperative period, with no obvious soakage of dressing. The arrow indicates that the swelling is reduced, and the brain is not tight; (B) Postoperative of lower limb cellulitis after debridement and fasciotomy in the early postoperative period. The dressing is soaked with exudates warranting frequent dressing and reassessment; (C) Bedspace examination shows that the patient is mechanically ventilated, having shock on dual vasopressors, analgesia infusion, and paralytic infusion that is stopped.



Figs. 2A to C: The postoperative site after emergency intra-abdominal surgery in different patients with bedspace. (A) The patient underwent laparotomy for complicated intra-abdominal sepsis. On inspection, the surgical wound is closed with a vacuum dressing and connected to a vacuum device. The abdomen is distended, and small ecchymosis is seen near the operated site. The patient is having triple lumen Foley catheter (solid black arrow) and intraabdominal pressure is monitored with a transducer placed at the iliac crest level (red circle); (B) This is a patient who was previously operated on for intra-abdominal surgery and had an enterocutaneous fistula and secondary infection. The figure shows the open abdomen with transparent coloplast dressing. The broken arrow shows pus from the open wound and bag in the sides drain bile-stained feculent material; (C) The bedspace examination of patient in Figure 1B shows having shock on vasopressors. The patient is on total parenteral nutrition (TPN) infusion.

- *Drains:* Passive corrugated drains or small active drains may be placed either at or near suture lines. Most of the time the drains are placed in the cavity of interest and often multiple drains are placed. The drain contents usually consist of plasma, blood cells, tissues, and drainage contents. The drains are serious (plasma, yellow), sanguineous (blood), serosanguineous (plasma and blood), and purulent. Drains should be examined frequently to check for their position, color, quantity, etc. *See* **Figure 3 and Table 4** for details.
- *Stoma:* Stoma margins—healthy/gangrene, retracted. Stoma is commonly seen in the abdomen and sometimes in the neck (post-transthoracic esophageal surgery). It is important to know the color of the contents and quantity.



Figs. 3A to I: Surgical drains. This is from a patient who underwent laparotomy for a complex intraabdominal infection and underwent relaparotomy for intraabdominal bleeding. The drain shown is of day 4 of the second laparotomy. The figure shows surgical drains hung in order (A to D). Drain A, C, and D are passive drains that drain by gravity and drain B is an active way of drainage if compressed or drains passively by gravity if not compressed (as shown in the figure). Note the drains are labeled (circled in drain A) for ease of identification. The arrow (drain A) shows the drain fluid is thick and purulent. In the drain tubes of drain C, the fluid is more liquid, serosanguineous; (E) Vacuum drain. It is an electronically operated device where a special material is placed in the open wound and a gentle suction is applied to it and it is draining pus; (F) Serous drainage, common in any postoperative patient; (G) The broken arrow indicates dark greenish-brown fluid (bile with peritoneal fluid) from a patient with an anastomotic leak; (H) Purulent drain fluid in a patient with intra-abdominal sepsis postpigtail insertion; (I) Patient with stoma bag placed in suture line draining fluid with enterocutaneous fistula in the late postoperative period.

Palpation

The palpation may start at the surgical site. It is good to rub your hands to make them warm. Our hands are cold, especially after applying hand rub. Make sure the patient is explained about the procedure. Check the following in sequence:

1. *Warmth:* Gently place the hand near the surgical site to feel the warmth.
2. *Tenderness:* Gently press the tissue and look for pain. Rebound tenderness—a sign of parietal peritoneum involvement in peritonitis. The signs may be blunt when the patient is under sedation or an analgesic infusion. When suspecting underlying collection, a gentle squeezing may help to identify discharges.
3. *Pulsations:* Palpation of swelling/local change in contour: Palpate to identify enlargement of organs (hepatosplenomegaly, lymph nodes). While palpating check for pulsations—aneurysmal swelling can be identified (in the abdomen).
4. *Ultrasonography (USG):* A vital extension of palpation in critically ill patients. It provides vital information in real time. It is useful in thoracoabdominal pathologies and deep-seated collections. Rapid diagnosis of vascular compromises.
5. *Per rectal examination:* Often we forget to do per rectal examination. Impacted stools are common in ICU patients. It guides us in providing enema to such patients. The per-rectal examination helps in the identification of gastrointestinal bleeding (blood-stained finger, malena).

Place the patients in a lateral position. Give a sedation bolus to make the patient comfortable; check the tubes,

TABLE 4: Characteristics and examinations of the surgical drain.

Domain	***Description***	***Normal***	***Abnormal***
Position of drain and surrounding skin	• Note where the inner end is placed, i.e., which cavity? • Check the drain—skin fixation site	Redness, warmth, and swelling at the drain exit site that is reducing with time is common	• An increase in redness, warmth, and swelling is abnormal and may indicate infection • Loose fixations drain migrations compared to the depth fixed is abnormal
Color of contents	• The drain color usually transforms from red to pink, then from light yellow to clear with a healing wound • The color may indicate the type of fluid, e.g., red (blood, serosanguinous)	• The drainage is slightly yellow or tinged with pink. It is usually clear • This is seen in the first few days of surgery	Any blood, pus, bile, or feces
Smell of contents	When in doubt, pour the drain fluid into a gauze and smell it. It will be foul smelling when purulent and bowel content will have a feculent odor. Otherwise, the drain fluids do not have an odor	Usually, odorless	Foul/feculent smell—gives a clue of bowel perforation, anastomotic leaks
Consistency of fluid	• Liquid/watery: Serous will be more like a thin liquid • Thick—pus, coagulum	Thin and watery	Thick and with particles
Patency of drain	Whenever there is minimal drainage, check for blockage	Patent and free flow	May be blocked with necrotic debris, coagulum, and pus
Quantity	• Always have a check on the quantity of drain • Sudden increase or decrease should trigger further evaluation • It is measured in routine	The quantity reduces with time and as the wound heals	• Any increase in quantity is abnormal if it is fresh blood, bile, or feces • The large volume is always abnormal unless expected

lines, and vitals. Apply lignocaine jelly. Gently insert the finger watching the patient's face. Rotate the finger to feel for any swelling, impacted feces, blood staining of finger, and anal sphincter tone.

Percussion

Percussion is commonly performed in the abdomen and thorax. It is useful to differentiate gas versus fluids. Tympanic note or loss of normal dullness near the liver surface may increase the suspicion of perforation. Due to the availability of USG, shifting dullness is not performed routinely in ICU patients. It is easy to identify a distended urinary bladder by simple palpation and percussion, complimented by USG.

Auscultation

Auscultation is performed to listen to bowel sounds, confirm naso-/orogastric tube position, and added sounds (crepitations) from the lungs. Listening for bruit—aorta, renal artery, and carotid are important.

Other System Examination

A focused systemic examination will give vital information on organ dysfunction and titration of organ support. Patients are at risk of postoperative, pulmonary complications, circulatory failure, arrhythmias, cognitive dysfunction, stroke, delirium, gastrointestinal dysfunction, ileus, etc. Below is a detailed examination of each system which is beyond the scope of this chapter and refer to other chapters for details.

Pain

Pain should be actively assessed in all patients after surgery. It is challenging to assess pain in patients on mechanical ventilators. The common scales adapted to assess pain are Critical Care Pain Observation Tool (CPOT) and Behavioral Pain Scale (BPS). A verbal pain scale can be used if the patient can communicate. A careful look at the patient's face and posture may give a clue about the pain. Pain may hamper respiratory exercises, delay extubation, and aggravate pulmonary complications, especially with thoracoabdominal surgeries.

Look: At the patient's face (restless and anxious) and posture (frequent flexion of upper and lower limbs). Use appropriate pictures/cards if feasible to elicit pain.

Feel: Palpate or gentle pressure to the operated area. (Wincing or grimacing to touch/palpation). Check the bandwidth if the epidural catheter is placed to check its effectiveness.

Listen: The patient may produce incomprehensible sounds/moan while in pain. It is good to listen to the patient when communicating.

Nowadays many patients receive either patient-controlled analgesia (intravenous or epidural). The first step is to look for the adequacy of pain control, and the functioning of the system (syringes and cartridges). The patients receiving the epidural infusions should be assessed for band width; the dermatomes of interest are adequately covered symmetrically.

Nutrition

Assessing nutritional status should be an integral part and the examination may not be complete if we do not examine the nutritional status of the patient after surgery.

Look: Built of the patient (recent weight); muscle wasting (temporalis, limb muscles, especially the bulk); oral cavity—signs of malnutrition—ulcers, cheilitis, dental caries, dry mouth, stomatitis, glossitis, candidiasis, etc.; and skin—dry, scales, and redness.

Feel: USG of muscles, e.g., quadriceps femoris thickness and circumference. Peripheral edema—hypoproteinemia

Cardiovascular

In short, adequacy of tissue, perfusion should be examined in patients with circulatory failure. Focused echocardiography and rapid cardiovascular examination (see other chapters for details) give vital pieces of information.

Respiratory System

Particular attention is to be given looking for restriction of chest wall/abdomen movements with respiration (due to pain, surgical dressings, pleural effusions/ascites after fluid infusions), basal atelectasis and hypoxia (thoracoabdominal surgeries), bradypnea with opioid analgesics, etc.

Gastrointestinal

Feed tolerance and bowel movements should be assessed daily in all patients. Nausea, vomiting, and paralytic ileus are common after any surgery and will resolve with time.

Central Nervous System

A quick assessment of the Glasgow Coma Scale, or FOUR score, depth of sedation and careful screening for delirium are important in patients after surgery. A detailed examination is warranted for neurosurgical patients and to a certain extent in patients who underwent cardiac surgery and are at risk of stroke.

SUMMARY

A focused, quick, and systematic examination is important in all patients postsurgery. The examination should be rapid when it is on demand. A quick look at the postoperative site, vital organ function, and bed space provides vital information. Nutritional status and pain should be assessed in all patients along with other systemic examinations. Critically ill patients may not show classical signs to elicit, and care should be given while examining such patients since the signs are often confounded by medications, organ failures, and support. The examinations should be supplemented by relevant focused sonographic assessment in appropriate settings.

SUGGESTED READING

1. Approach to the surgical patient. In: Doherty GM (Ed). Current Diagnosis and Treatment: Surgery, 14th edition. Jaypee Medical; 2015.
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3. Ramesh BA, Evans JT, BK J. (2023). Suction drains. [online] Available from https://www.ncbi.nlm.nih.gov/books/NBK557687/ [Last accessed March, 2024].
4. Schultz G, Tariq G, Harding K, Carville K, Romanelli M, Chadwick P, et al. (2019). WUWHS Consensus Document: Wound Exudate, Effective Assessment and Management. [online] Available from https://woundsinternational.com/world-union-resources/wuwhs-consensus-document-wound-exudate-effective-assessment-and-management/ [Last accessed March, 2024].

Chapter

19

Burns: Clinical Examination of the Critically Ill Patient

Indira Menon, Gayathri Priya Bindu Vanguri

ABSTRACT

All patients who sustain severe burn injuries should be ideally cared for in dedicated burns centers. As such centers are scarce, the onus of looking after these patients often falls on the intensive care units (ICUs) of hospitals.

It is important to identify the patient with burns who needs ICU admission. It is imperative to ensure the availability of certain facilities and equipment at the bedside to avoid delay and exposure of the patient to infection. Isolation practices and inclusion of a few specialty consultations is advisable. A secondary survey has to be performed in the ICU to accurately estimate the grade and severity of the burns. The history should be reviewed and the clinical examination by the intensivist should be a detailed one with focus on airway, breathing, circulation, and bony injuries. Video laryngoscopy and ultrasound to monitor airway congestion could avoid unwarranted endotracheal intubation. Fluid management remains the cornerstone of treatment with emphasis on euvolemia. "Fluid creep" has been associated with complications and should be avoided. Daily dressings with saline or silver sulfasalazine/mupirocin ointment is important. Early surgical excision of dead tissue promotes faster recovery. Prophylactic antibiotics are not recommended. The main complications of the burns are secondary infection, compartment syndromes, and acute kidney injury. The intensivist should ensure that psychological support is provided to both the patient and the family members.

INTRODUCTION

Injuries sustained due to burns are a worldwide occurrence causing devastating consequences. The World Health Organization (WHO) estimates that 11 million people per year worldwide sustain severe burn injuries and 8 million have disability adjusted life years (DALYs). In India, 1.5 million people have DALYs secondary to burns. In 2019, there were 23,000 fire-related deaths in our country, which is 20% of the global burden.

Burns patients in India are primarily in the 15–49 years age group and women outnumber men by three times. Financial burden, loss of jobs, and social discrimination have unmeasured impact on the survivor.

There are only 67 dedicated burns units in our country, and they are unable to cater to the large load of cases.

Therefore, it is vital that all intensive care units (ICUs) be equipped to handle the critically ill burns patient.

Knowledge of the pathophysiology of burns is key to managing the patient. The initial assessment, intensive monitoring and management have a huge impact on the outcomes of these patients.

The arrival of a seriously injured burns patient can cause a stir in any ICU. This chapter aims at providing an overview of the clinical and practical approach to the care of these patients and will highlight the following aspects:

- Criteria for ICU admission
- History and symptomatology review
- Preparedness of the ICU to receive burns patient
- Clinical examination—assessment of severity (surface area/depth)
- Clinical impact of the injury (airway/breathing/circulation/systemic head to toe examination/laboratory tests)
- Management (medical/surgical/complications)

APPROACH TO THE CRITICALLY ILL BURNS PATIENT

Case scenario: *Mrs S was brought to the emergency room (ER) with a history of having sustained severe burns in her home while cooking on a gas stove. The fire fighters found her on the floor and rushed her to the hospital. In the ER, initial resuscitation was done. Her BP was 140/90 mm Hg,* *heart rate was 120 beats/min,* SpO_2 *98% on venturi mask, and* FiO_2 *0.5. Intravenous (IV) fluids were started, the percentage of burns was assessed as 50%, and a bed was arranged in the ICU.*

Which burns patient require admission to the ICU?

- Adults with >20% burns
- Children with >10% burns
- Elderly >60 years of age
- Persons with inhalational injury
- Persons in shock
- Persons with organ dysfunction, for example, altered mental status/renal failure
- Persons with circumferential burns that could compromise respiration or limb circulation
- Persons displaying compartment syndrome—either limb or abdominal
- Associated with major trauma

History and Symptomatology

It is important to verify certain aspects of the situation of the burns injury as it affects the management and prognosis.

- Did the burns occur in a *closed space or open air*?—carbon monoxide poisoning is more likely to be associated with closed spaces burns.
- Any *history of fall or evidence of fractures* on primary survey?
- What kind of *heat source* was it?—flames, scald, electric? The temperature varies according to source and the extent of skin injury depends on the temperature it is exposed to. Some burns such as electric and lightning are associated with cardiac arrhythmias and fractures of vertebrae/long bones.
- What is the *type of burns*?—superficial would be painful and deep would not have much pain.
- Has any *pain medication* been administered? This could affect the sensorium and respiratory function.
- Is there *involvement of the face and neck*? The chance of respiratory compromise is higher and airway management may be difficult.
- Does the patient have any *breathing difficulty/stridor*?
- Is the *patient intubated*? If so, is the endotracheal (ET) tube cuffed and uncut? The ET tube should not be cut as the subsequent facial swelling that may develop could cause difficulty with the length.
- What *IV lines* have been secured? At least two peripheral large bore lines should be secured, preferably through unaffected skin. Very often, central venous access via the least affected skin will need to be obtained.
- Have the *ER staff washed the patient's skin* with 12–15°C temperature clean water for 2–3 minutes?

What arrangements should the ICU staff make in order to receive a burns patient?

- Isolation room—positive pressure isolation and temperature (28–34°C) control necessary
- Intubation tray with equipment for difficult airway management including a video bronchoscope/laryngoscope
- Central line tray
- Arterial line insertion facilities
- Urinary catheterization facility
- Strict aseptic precautions must be followed.
- Additional help may be required from other specialties such as ear, nose, and throat (ENT)/orthopedics/plastic surgery.

Mrs S was brought to the ICU. She was conscious with Glasgow Coma Scale (GCS) 15/15. Her respiratory rate was 24 breaths/min, SpO_2 *90%, pulse 120 beats/min, and BP 90/70 mm Hg. Her body weight was 60 kg and height 160 cm. She had burns over her face, neck, abdomen, and thighs. Her back had been spared.*

General Examination

The intensivist immediately performed a detailed general examination, which includes assessment of the airway and establishing adequate circulation. The secondary survey should reassess the percent of burns and a head-to-toe examination of the patient must be conducted to document all injuries sustained. Primary surveys often overestimate the percentage of burns.

Assessment of Severity of Burns

Based on Surface Area

- *Wallace's "Rule of Nines"* is a simple method of calculating total body surface area (TBSA) affected in burns patients **(Fig. 1)**.

In adult patients, head and neck (9%), anterior chest and abdomen (18%), posterior chest and abdomen (18%), each upper extremity (9%), each lower extremity (18%), and perineum (1%).

Wallace's Rule of Nines is an easy method to estimate the extent of burns. Mrs S had involvement of head and neck, abdomen and chest anteriorly, and upper thighs and arms anteriorly. The total burn area was therefore approximately 45%.

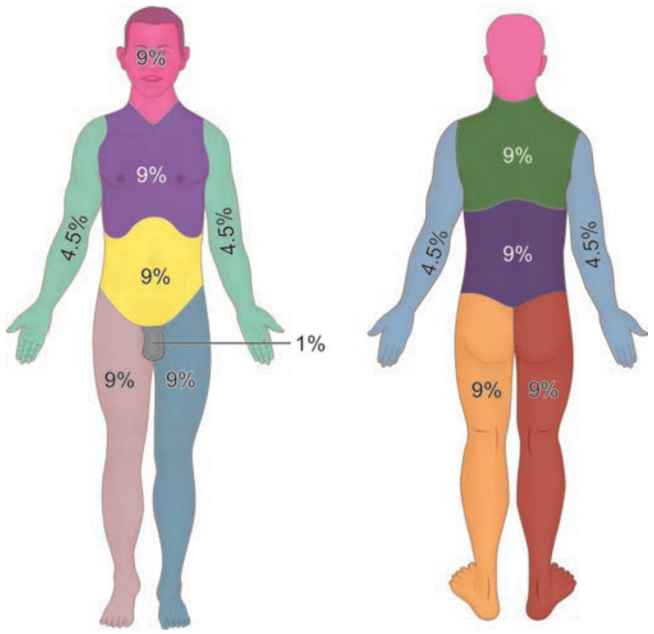


Fig. 1: Wallace's "Rule of Nines" in adults.
Source: Adapted by I Menon based on image courtesy of American Burns Association. Determining Total Body Surface Area. [online] Available from https://www.health.state.mn.us/communities/ep/surge/burn/tbsa.html. [Last accessed April, 2024].

The other methods of measuring the TBSA of burns are the Lund–Browder method and the patient's palm method.

- *Lund and Browder chart* ***(Table 1)****:* Most accurate, accounts for body proportions by age group.

It is important to note that in this chart, only second degree and third degree burns are counted as part of the total TBSA burn percent.

- *"Patient's Palm" method*—the patient's palm is roughly equivalent to 1% of the TBSA.

  The palm of the patient who has sustained burns is considered to be 1% of the patient's TBSA. Therefore, the percentage of burns is estimated using the patient's own palm.

Severity Based on Depth

Degree of burns is assessed based on the depth of burns **(Table 2)**.

Assessment of Clinical Impact of Burns

Airway and Breathing

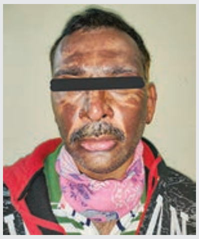
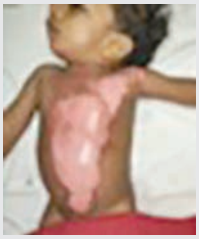
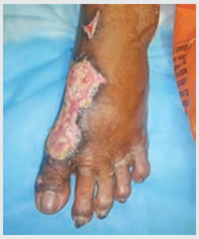
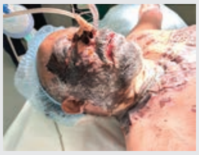
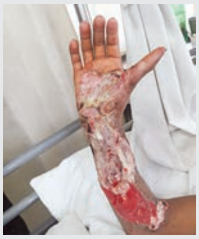
- Look for burns around the nose, oropharynx, and neck.
- Are there any blackish deposits in the oropharynx?
- Look for stridor or any expiratory wheeze.

TABLE 1: Lund–Browder chart for assessing surface area of burns.

Area	*Time of birth—1 year*	*1–4 years*	*5–9 years*	*10–14 years*	*15 years*	*Adult*	*2′*	*3′*	*Total*
Head	9	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	13	13	13	13	13			
Left upper arm	4	4	4	4	4	4			
Right upper arm	4	4	4	4	4	4			
Left lower arm	3	3	3	3	3	3			
Right lower arm	3	3	3	3	3	3			
Left hand	2.5	2.5	2.5	2.5	2.5	2.5			
Right hand	2.5	2.5	2.5	2.5	2.5	2.5			
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
Left thigh	5.5	5.5	5.5	5.5	5.5	5.5			
Right thigh	5.5	5.5	5.5	5.5	5.5	5.5			
Left calf region (Lower leg)	5	5	5	5	5	5			
Right calf region (Lower leg)	5	5	5	5	5	5			
Left foot	3.5	3.5	3.5	3.5	3.5	3.5			
Right foot	3.5	3.5	3.5	3.5	3.5	3.5			
						Total:			

Source: Bittner EA, Shank E, Woodson L, Martyn JA. Acute and perioperative care of the burn-injured patient. Anesthesiology. 2015;122(2):448-64.

TABLE 2: Summarization of association of degree of burns and management.

Degree of burns	***Depth of burns***	***Appearance***	***Healing time***	***Management***	***Examples***
First degree (Superficial)	Epidermis, e.g., sunburns	Hyperemic or hyperpigmented (in darker individuals) tender, no blisters	5–7 days	*Conservative:* Protective dressing, analgesia, intravenous fluids for extensive injuries	
Second degree (Superficial partial-thickness burns)	Epidermis and papillary dermis	• Characteristic features—blistering and blanching on pressure • Painful due to exposed nerve endings	7–14 days	Moist occlusive dressings, topical antimicrobials, analgesics, antiedema measures, physiotherapy	
Second degree (Deep partial-thickness burns)	Entire thickness of skin including reticular dermis	No blanching, dry with near total destruction of dermal glands and hair follicles	3–5 weeks	*Conservative:* Excision and grafting	
Third degree (Full thickness burns)	Dermis, adnexal structures, and subcutaneous adipose tissue	• Hard, leathery, insensate with no capillary return. Often, thrombosed vessels can be seen under the skin • Circumferential full thickness burns impair the circulation	Does not heal by primary intention	Escharotomy Excision and grafting	
Fourth degree burns	Involves underlying muscle, bone, or tendon	Black and charred eschar	Does not heal by primary intention	Escharotomy, fasciotomy, debridement and flap cover, amputation when necessary	

Source: Pictures taken by B Vanguri. Table based on Jackson's Criteria of Burns.

As Mrs S had sustained burns to the face and neck, it is necessary to secure the airway early before the inflammatory edema sets in. However, the practice of prophylactic intubation is not recommended routinely, even in patients with inhalation syndrome or cervical burns. Although Venus B et al. did recommend prophylactic intubation in all patients with inhalation injury in order to decrease mortality, intubation carries its own risks of

TABLE 3: Guidelines for intubating burns patients according to the ABA criteria and Denver criteria.

ABA criteria	***Denver criteria***
Signs of airway obstruction—hoarseness, stridor, accessory muscle use, and sternal retraction	Any of the ABA criteria listed with two more: singed facial or nasal hair/smoke inhalation
Extent of burn >40% TBSA	
Extensive facial burns—deep dermal or full thickness depth	
Dysphagia	
Intraoral burns	
Signs of respiratory compromise—respiratory fatigue, hypoxia, and poor ventilation	
Reduced LOC with loss of protective reflexes	
Anticipated transfer of patient with major burn to burn center without qualified medical professional to intubate en route	
Significant risk of edema, which may compromise airway	

(ABA: American Burns Association; LOC: level of consciousness; TBSA: total body surface area)

Source: Based on ABA and Denver Criteria.

1. American Burn Association. Advanced Burn Life Support Provider Manual 2018 update. J Burn Care Res; 2018.
2. Allorto, Nikki, et al. "ISBI Practice Guidelines for Burn Care, Part 2." Burns. 2018;44(7):1617-706. https://doi.org/10.1016/j.burns.2018.09.012.

morbidity—need for sedation, tracheal injury, mechanical ventilation, and ventilator-associated pneumonia (VAP).

Many studies since then have advised against unnecessary intubations.

In 2015, Romanowski et al. found that 40% of the patients were extubated within 48 hours, suggesting that one-third of intubations in burns patients were unwarranted. Of course, a late airway approach could be catastrophic, and therefore the 2016 guidelines from the International Society for Burn Injury recommend observation and monitoring with intubation/tracheostomy if airway patency is endangered.

Immediate securing of airway is indicated if patient has a low GCS <8, stridor present, respiratory fatigue, inability to clear secretions, >40% TBSA, or hypoxia needing supplemental oxygen. The final decision however rests with the treating physician and the expertise available for difficult airway intubation.

Most burns centers use the American Burns Association (ABA) or the Denver criteria to intubate patients **(Table 3)**. Video laryngoscopy, video bronchoscopy, and airway ultrasound are becoming increasingly popular to serially monitor the development of congestion and edema of the airways and to allow for intervention at the optimal time. These procedures are performed 2 hourly in patients at high risk **(Figs. 2 to 4)**.

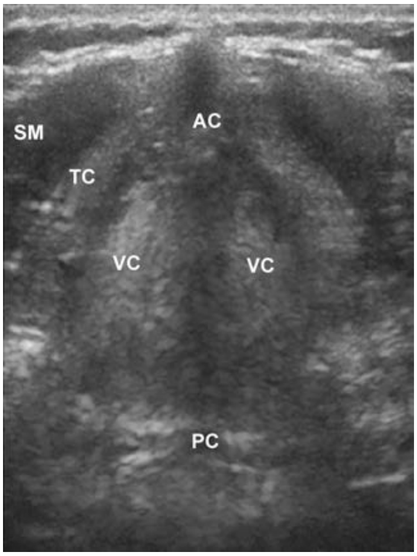


Fig. 2: Ultrasound imaging of normal larynx. (AC: anterior commissure; PC: posterior commissure; SM: strap muscles; TC: thyroid cartilage; VC: vocal cords)

Source: Kameda T, Fujita M. Point-of-care ultrasound detection of tracheal edema caused by smoke inhalation. Crit Ultrasound J. 2014;6(Suppl 1):A26. Reprints and Permissions.

Nebulization with 10,000 units of heparin and 600 mg N-Acetylcysteine every 6 hours may help reduce the lower airway edema and inflammation and could be used as adjuncts **(Flowchart 1)**.

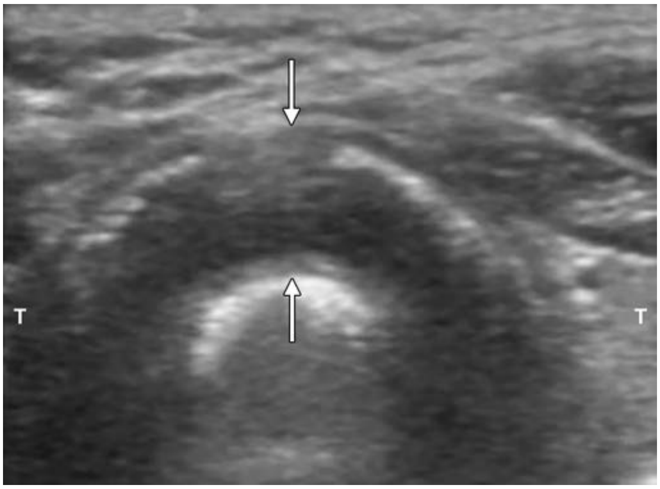


Fig. 3: Ultrasound imaging of normal trachea. (T: thyroid gland)
Source: Kameda T, Fujita M. Point-of-care ultrasound detection of tracheal edema caused by smoke inhalation. Crit Ultrasound J. 2014;6(Suppl 1):A26. Reprints and Permissions.

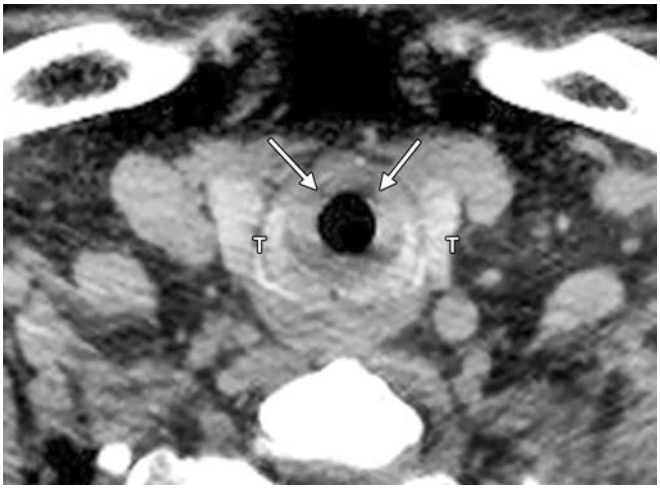
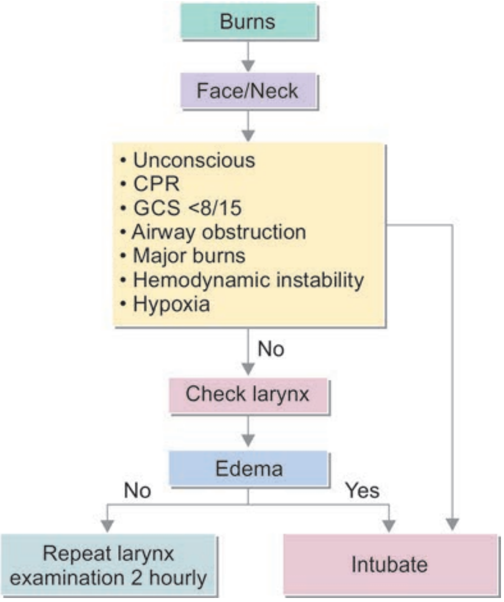


Fig. 4: Ultrasound image of tracheal edema. (T: thyroid gland)
Source: Kameda T, Fujita M. Point-of-care ultrasound detection of tracheal edema caused by smoke inhalation. Crit Ul-trasound J. 2014;6(Suppl 1):A26. Reprints and Permissions.

While performing intubation on a burns' patient, the intensivist has to anticipate a difficult airway scenario and the most experienced doctor on duty should perform the intubation.

- Rapid sequence intubation was performed on Mrs S and suxamethonium for muscle relaxation was used as it was <48 hours after the injury.
- The ET tube should be cuffed and should not be cut as edema that develops later could compromise the tube's length.

Flowchart 1: Immediate assessment of airway in burns patient.



(CPR: cardiopulmonary resuscitation; GCS: Glasgow Coma Scale)
Source: Created by I Menon.

The role of tracheostomy in the critical burns patient is controversial as most of them could be weaned off the ventilator without tracheostomy. Mourela et al. found no difference in mortality between burns patients who underwent tracheostomy and those who were intubated. They noted that the tracheostomy patients had longer hospital stay and ventilatory days but there was no difference in VAP between the 2 groups. The study by Aggarwal et al. noted increased VAP in the tracheostomy group as compared with those that were intubated.

Once the patient is placed on a ventilator, lung protective ventilation with low tidal volumes to ensure driving pressure <15 mm Hg is initiated.

- 100% O_2 may be needed for 6–12 hours if carbon monoxide poisoning is suspected.
- Look for possible cyanide toxicity—exposure to burning nitrogen containing material causes inhibition of cell metabolism leading to shock and coma. These patients are given 5 g of hydroxocobalamin over 15 minutes intravenously.
- Exposure to hydrofluoric acid causes severe tissue damage and metabolic acidosis for which local calcium gel or IV calcium gluconate can be used.

Circulation

Hemodynamic stability is the mainstay of managing the burns patient. Release of cytokines, leukotrienes, and prostaglandins could lead to disruption of the microcirculation, hypovolemia, myocardial dysfunction, and depression of the vascular tone.

Initially, severe hypovolemic shock due to capillary leakage causes decrease in cardiac output (CO) and increase in systemic vascular resistance (SVR). The reduction in CO is not only due to hypovolemia but also due to increased afterload and decreased contractility secondary to the inflammatory mediators and the impaired calcium transport in the myocardium. However, subsequently, after 2 hours the CO could increase and the SVR should decrease due to inflammation.

Edema in the burn region becomes maximal after 24 hours.

Therefore, the combined effect of systemic microvascular damage, massive edema with impaired lymphatic drainage, and reduced CO overpowers optimal circulatory functions and hampers tissue oxygenation.

Clinical assessment of volume loss such as tachycardia, hypotension, and high hematocrit is an important indicator of optimal circulation.

An international survey revealed that doctors managing burns patients used different hemodynamic assessment modalities with 40% preferring CO monitoring, 40% pulse pressure variation/stroke volume variation, 60% serum lactate, and 75% relying on urine output monitoring.

In the burns patient, central venous pressure (CVP) and urine output are more reliable than the sophisticated methods of fluid assessment.

An invasive arterial line may be needed in all patients with extensive burns for accuracy of measurement. In addition, the application of a BP cuff could be difficult.

Although there are many formula for fluid calculation in the burns patient, the half Parkland formula or the Modified Brooke formula is the most accepted at present.

Our patient was calculated to have 5,400 mL fluid requirement. We administered 50% of the volume in 8 hours and the rest over 16 hours **(Table 4)**.

TABLE 4: Complications associated with volume resuscitation of the burns patient.

Less volume	***Excess volume***
Decreased tissue perfusion	Edema
Splanchnic ischemia (mesenteric)	Intra-abdominal hypertension
Renal failure	Respiratory failure
Multiorgan failure	Renal failure
	Multiorgan failure/impaired healing

Source:

1. Boehm D, Menke H. A History of Fluid Management—From "One Size Fits All" to an Individualized Fluid Therapy in Burn Resuscitation. Medicina. 2021;57(2):187. https://doi.org/10.3390/medicina57020187
2. Mph, Griffin L. Davis Md. Volume Resuscitation: Background, Indications, Contraindications. emedicine.medscape.com/article/2049105-overview?&icd=login_success_email_match_fpf.

Keeping in mind that higher cumulative fluid balance is associated with higher mortality and increased need for renal replacement therapy, the administration of fluid (crystalloid ringer lactate) has to be individualized and a urine output of 0.5 mL/kg/h to 1 mL/kg/h has to be ensured.

It is important to stress that excessive fluid resuscitation can cause the *"fluid creep",* which leads to increased abdominal compartment syndrome, intestinal wall edema, acute respiratory distress syndrome (ARDS), pulmonary edema, and prolonged mechanical ventilation. Therefore, one should avoid urine output >1 mL/kg/h, abdominal pressures >20 mm Hg, development of acute kidney injury (AKI)>, visceral ischemia, and lowering PaO_2/FiO_2 ratio, which are signs of excess hydration.

Burns experts advise "permissive hypovolemia" with the goal being to stay "just on the dry side of euvolemia". This requires hourly monitoring and evaluation.

The modified Brookes (or half Parkland) formula is recommended for fluid administration and the goals of volume resuscitation in burns injury are as follows **(Box 1)**:

- Mean arterial blood pressure ≥60 mm Hg
- Urine output >0.5 mL/kg/h
- Normal pH and base excess
- Normal lactates

Ideally, the patient should return to normal body weight by 7–10 days.

Systemic Examination

Once the airway and circulation have been addressed, a detailed head-to-toe survey of the patient is done.

Eyes: Should be examined for injury/swelling and fluorescein staining may be needed to identify epithelial

BOX 1: Formula for fluid administration in burns patients.

1. *Rule of 10:*
   %TBSA × 10 = Initial mL/h
   If patient weighs >80 kg, add 100 mL/h for every 10 kg above 80 kg
2. *Parkland formula:*
   %TBSA × 4 mL/kg: Give half the volume in 8 hours and the remaining over the next 16 hours
3. *Half Parkland formula = 2 mL/kg/% TBSA*
4. *Modified Brooke formula:*
   %TBSA × 2 mL/kg: Give half over 8 hours and remaining over 16 hours.

Source: Chung KK, Salinas J, Renz EM, Alvarado RA, King BT, Barillo DJ, et al. Simple derivation of the initial fluid rate for the resuscitation of severely burned adult combat casualties: in silico validation of the rule of 10. J Trauma. 2010;:69 (Suppl 1):S49-54. doi: 10.1097/TA.0b013e3181e425f1.

injury. Lubricants should be applied. Tarsorrhaphy is not needed often.

Mrs S had corneal opacification of the left eye with visual impairment.

Cardiac and respiratory system: Monitoring for arrythmias. Bedside ultrasound to assess myocardial dysfunction. Chest examination for evidence of pulmonary edema, ARDS, aspiration pneumonia, and VAP. This could be confirmed with the help of lung ultrasonography.

Vasculature, extremities, and spine: Assess for fractures. Monitor distal blood supply with capillary refill time and doppler ultrasound, especially in patients with circumferential burns of limbs.

Abdomen: Extensive burns over the abdomen require monitoring of intra-abdominal pressures.

Mrs S did not develop abdominal compartment syndrome nor any gastrointestinal (GI) bleeding. She was given nasogastric tube feeding and stress ulcer prophylaxis.

Genitourinary tract: Urinary catheterization is needed to monitor hourly urine output.

Neurological: Findings at admission should be documented as the sensorium could deteriorate later due to metabolic factors and medications administered.

Laboratory Investigations

- *What are the initial blood samples that need to be sent?*
  - Once the examination and assessment are over, several investigations need to be carried out.
  - Blood counts including hematocrit, serum electrolytes, BUN (blood, urea, nitrogen) and creatinine, calcium, magnesium, blood sugars, creatinine kinase, liver function tests, and arterial blood gas analysis (lactates, base deficit, and P/f ratio) should be assessed. The hematocrit, lactate, and base excess are useful adjuvants to assess fluid management.
  - Blood cultures should be sent after the initial cleaning of the patient.
  - *Skin and wound biopsy cultures should be sent every week* and are more reliable than peripheral cultures to identify the infecting organism.
- *What were the initial medications started for Mrs S?*

Management

Sedation and Analgesia

Providing pain and anxiety relief to these patients is a major concern for the treating intensivist and the patient was regularly assessed for the need for analgesia especially during the daily surgical dressing change.

- Since she was being invasively ventilated, she was given midazolam and fentanyl infusion. Alternative medication could be ketamine infusion 1 mg/kg/day or dexmedetomidine 0.2–0.7 µg/kg/h.
- Injection paracetamol 15 mg/kg/24 h in four divided doses could also be used if patient was not being ventilated along with low-dose morphine 0.15 mg/day.

The need for analgesia is more in the initial days and tends to decrease with time.

No role for prophylactic antibiotics.

How soon can nutrition be initiated for this patient?
Burns is a hypermetabolic state that could last up to a year after the event. Nutrition can be initiated within 8–12 hours of burns and enteral nutrition is the preferred mode. Adequate calorie intake ensures quicker recovery. Prevention of lean body mass loss by maintaining a warm environment and using beta blockers/insulin is also recommended.

Calorie requirement in patients with burns is usually calculated using the Toronto equation:

$$\text{Total EE} = -4.343 + (10.5 \times \%\text{BSA}) + (0.23 \times \text{CI}) + (0.84 \times \text{REE H-B}) + (114 \times T^\circ\text{C}) - (4.5 \times \text{days})$$

where CI = calorie intake, T = fever, REE = resting energy expenditure from the Harris and Benedict equation, BSA = body surface area

Burns patients need 2.5 g/kg/day protein and 1.5–1.7 times the calories calculated by the basal metabolic rate, or 1.3 to 1.5 times the measured energy requirement at rest. Lipids should be <1.5 g/kg/day.

Parenteral nutrition is only advocated if 60% of the daily calories cannot be given enterally for >4 consecutive days.

Vitamin C in a high dose of 66 mg/kg/h in the first 24 hours reduces the fluid requirement by 40% in a study by Tanaka. Vitamin D supplementation and trace elements selenium, zinc, and copper should be given.

Mrs S underwent daily saline dressing and debridement in the ICU. Some centers use specific ointments such as 1% silver sulfadiazine, mupirocin, or nanocrystalline silver. On day 3, it was noticed that the left leg was looking paler and the left dorsalis pedis pulse was weak.

She was immediately taken up for escharotomy and fasciotomy in view of the compartment syndrome. Temporary autograft skin grafting was done.

Escharotomies are full thickness incisions made over circumferential deep partial and full thickness burns to release the constriction caused by the inelastic skin. They are done for third-degree burns **(Figs. 5A and B)**.

Fasciotomy is mainly done for compartment syndromes where the incision is made until the fascia in order to release the pressure on the swollen muscles. Patients with electric burns often need fasciotomies **(Figs. 6A and B)**.

Approach to wound care varies between centers.

Autografts are the gold standard of wound care, and they provide a covering, which reduces fluid protein and heat loss, while also granting protection against microbial colonization. If the patient does not have adequate normal skin for autografts, then allografts from cadaveric origin can be used. These allografts are either fresh or preserved in glycerol or via cryoprecipitation.

Availability of skin banks could facilitate the procurement of this cadaveric skin.

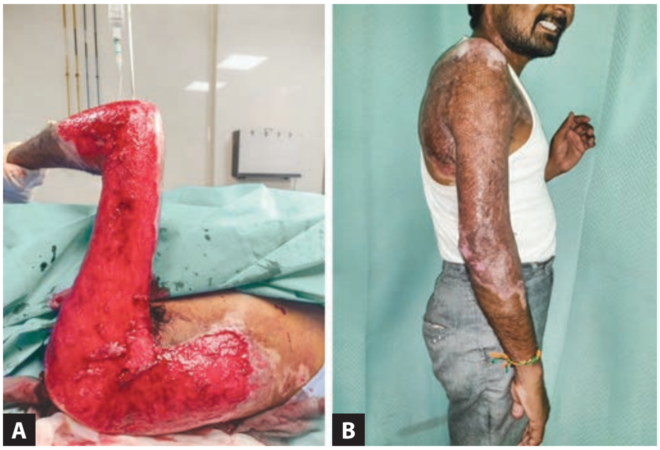
Though expensive, biosynthetic skin substitutes such as Integra and acellular dermal matrix could be used on functionally important areas to improve healing. Cultured keratinocytes have also been sprayed over the autograft donor sites to facilitate wound healing.

During the escharotomy, there was significant bleeding and her Hb dropped to 6.5 g%. The prothrombin time (PT)/ international normalized ratio (INR) was 1.5. She was transfused 1 packed red blood cell (PRBC) following which her Hb increased to 7.5 g%.

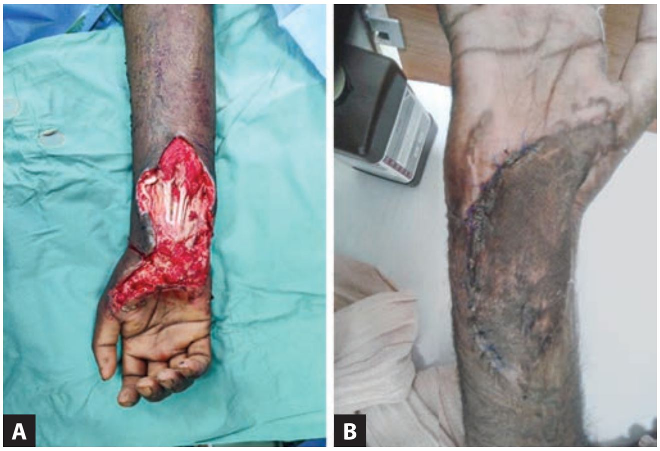
Wound debridement and flap cover: Previously most surgeons performed serial debridement of the dead tissue followed by skin grafting after 3–4 weeks. This led to hypertrophic scarring and repeated graft rejection.

However, Jackson (1960) and Janzekovic (1970) proposed early excision of the burn tissue to remove all devitalized tissues and demonstrated less infection, faster recovery, and decreased mortality in their patients.

There are various techniques to excise dead burn tissue.



Figs. 5A and B: (A) Wounds over right upper limb and posterior trunk following excision of third-degree burns; and (B) Follow-up picture showing well settled skin graft at 1 year.
Courtesy: B Vanguri.



Figs. 6A and B: (A) Distal volar forearm defect exposing tendons and nerves and (B) Follow-up picture is taken 6 months after flap surgery.
Courtesy: B Vanguri.

Most burn wounds are *excised tangentially* in order to preserve the contours, promote faster healing, and reduce hospital stay. However, this technique could lead to significant blood loss.

Fascial excision, though an easier alternative with less blood loss, could result in contour loss and lymphoedema. As a result, it is only done in life-threatening situations, especially in fungal infections.

Amputation is applicable in unsalvageable limbs, in very deep burns, and electrocutions.

On day 7, Mrs S developed hypotension. Her BP was 70/50 mm Hg, heart rate 130 beats/min, temperature 102°F, and FiO_2 *requirement increased from 40% to 80%. The nurse noted excessive ET secretions and feed intolerance. Her urine output was 0.5 mL/kg/h. The skin over the abdomen had yellowish discharge.*

She had secondary infection with the possible source of sepsis being either skin or VAP. We sent cultures of the ET secretions, paired blood cultures, and skin biopsy culture, and started her on noradrenaline infusion. Piperacillin/tazobactam was initiated as per the antibiotic policy. Echocardiogram was repeated to rule out cardiac vegetations. The fever and shock persisted and on day 9, her central line and urinary catheters were changed. The microbiological results showed growth of Klebsiella in her blood and skin biopsy cultures, whereas the ET secretions revealed heavy growth of Acinetobacter. She was in gram-negative sepsis and her antibiotics were escalated to meropenem and colistin as per the sensitivity.

Sepsis in the burns patient can be difficult to identify in the presence of the severe systemic inflammation findings.

Typically, fever >39°C, increased tachycardia, low blood sugars, poor gastric emptying, and raised procalcitonin levels could help identify the onset of sepsis.

There are some sets of criteria to detect sepsis onset in patients with severe burns.

Different Criteria for Sepsis

The criteria for identifying sepsis in burns patients are presented in **Table 5**.

Common Microorganisms Causing Infection in Burns Patients

Common organisms identified in burns-related sepsis are presented in **Table 6**.

During the course of her treatment with meropenem and colistin, Mrs S was found to have a drop in urine output to <0.3 mL/kg/h for 4 consecutive hours. Arterial blood gas analysis did not reveal significant hyperkalemia or metabolic acidosis. Her hemodynamic parameters were checked and optimized, and a fluid challenge with Ringer Lactate was administered to her. She responded to it and did not need any renal replacement therapy.

TABLE 5: Criteria available for identifying sepsis in burns patients.

ABA sepsis criteria	***Mann-Salinas et al. Novel burn-specific sepsis predictors***	***(Sepsis-3) Consensus definition for sepsis***
At least one or more of the following: 1. Temperature >39°C or <36.5°C 2. Progressive tachycardia (>110 bpm) 3. Progressive tachypnoea 4. Thrombocytopenia 5. Hyperglycemia 6. Inability to continue enteral feedings 24 hours	1. Tachycardia >130 bpm 2. MAP <60 mm Hg 3. Base deficit <–6 mEq/L 4. Hypothermia <36°C 5. Use of vasoactive medications 6. Hyperglycemia >150 mg/dL	*Sepsis:* Suspected or documented infection and an acute increase of ≥2 SOFA points or a qSOFA ≥2 1. Altered mental status (Glasgow Coma Scale <13) 2. Systolic blood pressure ≤100 mm Hg 3. Respiratory rate ≥22 breaths/min
Infection, at least one of the below: 1. Positive culture 2. Pathologic tissue source identified 3. Clinical response to antimicrobial agents AND at least three of the following predictors:		*SOFA variables:* 1. PaO_2/FiO_2 ratio 2. Vasopressor requirement 3. Serum creatinine or urine output 4. Platelet count 5. Bilirubin 6. Mean arterial pressure 7. Glasgow Coma Scale

Contd...

Contd...

ABA sepsis criteria ***At least one or more of the following:***	***Mann-Salinas et al. Novel burn-specific sepsis predictors***	***(Sepsis-3) Consensus definition for sepsis***
		Septic shock: 1. Septic shock predictors (sepsis and both predictors) 2. Vasopressors required to maintain MAP >65 mm Hg 3. Lactate >2 mmol/L (after adequate fluid resuscitation)

(ABA: American Burns Association; bpm: beats per minute; FiO_2: fraction of inspired oxygen; MAP: mean arterial pressure; PaO_2: partial pressure of arterial oxygen; qSOFA: quick SOFA; SOFA: Sequential Organ Failure Assessment)

Source: Yan J, Hill WF, Rehou S, Pinto R, Shahrokhi S, Jeschke MG. Sepsis criteria versus clinical diagnosis of sepsis in burn patients: A validation of current sepsis scores. Surgery. 2018;164(6):1241-5.

TABLE 6: Organisms identified in burns-related sepsis.

Group	***Species***
Gram-positive organisms	• *Staphylococcus aureus* • Methicillin-resistant *Staphylococcus aureus* • Coagulase-negative staphylococci
Gram-negative organisms	• *Pseudomonas aeruginosa* • *Escherichia coli* • *Klebsiella pneumoniae*
Fungi	• *Candida* spp. • *Aspergillus* spp. • *Mucor* spp.
Viruses	Herpes simplex virus Cytomegalovirus Varicella-zoster virus

Source: Zhang P, Zou B, Liou Y, and Huang C. The pathogenesis and diagnosis of sepsis post burn injury. Burns Trauma. 2021;9:tkaa047.

Acute kidney injury is a significant complication of burns injury with an incidence of 30%. AKI could occur early (0–3 days) or late (4–14 days). AKI has a multifactorial cause and could increase mortality to 80%. Renal replacement therapy is needed in 12–13% burns patients with AKI and progression to chronic kidney disease (CKD) is known to occur especially in those who need dialysis.

Helantera et al. in 2016 opined that burns accelerated the development of end-stage renal disease (ESRD) in patients with CKD. This was corroborated by Duan et al in 2021.

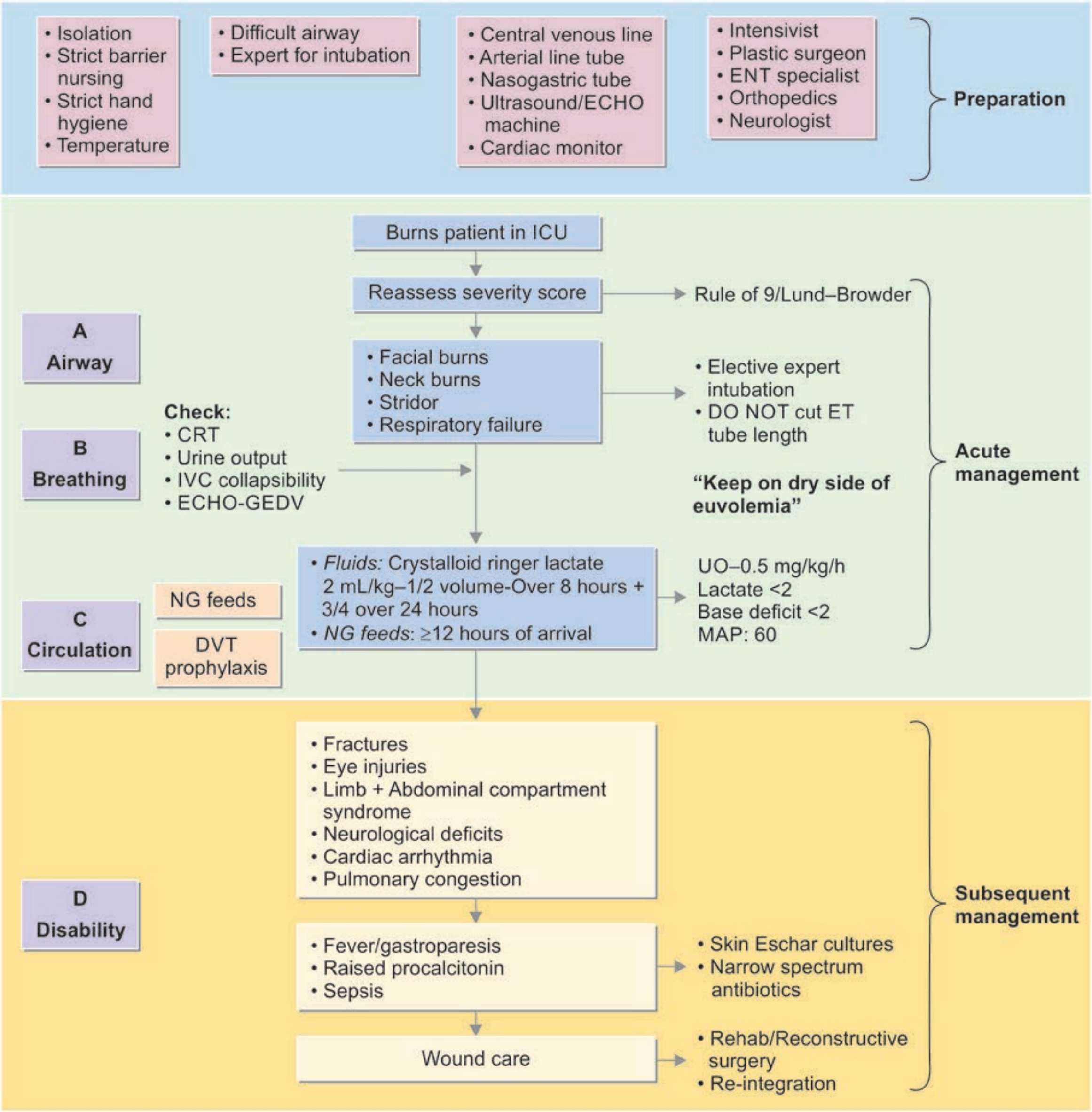
Her condition stabilized by day 20 and she could be extubated.

Mrs S was shifted to the isolation ward after 3 days of extubation and from then onward her recovery was rapid. She underwent multiple escharotomies and reconstruction procedures. She lost vision in one eye, but has managed to rebuild her life with the support of her family and with regular counselling sessions.

KEY POINTS

- There are very few dedicated burns centers in India. The challenge of managing the critically ill burns patient falls on ICUs.
- Secondary survey of the patient is imperative in the ICU, as the severity of burns is often overestimated in the emergency room.
- Airway management is different in the burns patient.
- Fluid resuscitation is the cornerstone of care, but "permissive hypovolemia" is the goal.
- Prophylactic antibiotics are not recommended.
- Nutrition is key to recovery.
- Wound excision and escharotomy are performed within the first week.
- Rehabilitation begins on day 1.
- Care of the burns patient is a multispecialty effort.

Flowchart 2: Summarizing overall management of the critically ill burns patient.


• Isolation
• Strict barrier nursing
• Strict hand hygiene
• Temperature
• Difficult airway
• Expert for intubation
• Central venous line
• Arterial line tube
• Nasogastric tube
• Ultrasound/ECHO machine
• Cardiac monitor
• Intensivist
• Plastic surgeon
• ENT specialist
• Orthopedics
• Neurologist
Preparation
Burns patient in ICU
Reassess severity score
Rule of 9/Lund–Browder
A
Airway
• Facial burns
• Neck burns
• Stridor
• Respiratory failure
• Elective expert intubation
• DO NOT cut ET tube length
B
Breathing
Check:
• CRT
• Urine output
• IVC collapsibility
• ECHO-GEDV
"Keep on dry side of euvolemia"
Acute management
C
Circulation
NG feeds
DVT prophylaxis
• Fluids: Crystalloid ringer lactate 2 mL/kg–1/2 volume-Over 8 hours + 3/4 over 24 hours
• NG feeds: ≥12 hours of arrival
UO–0.5 mg/kg/h
Lactate <2
Base deficit <2
MAP: 60
• Fractures
• Eye injuries
• Limb + Abdominal compartment syndrome
• Neurological deficits
• Cardiac arrhythmia
• Pulmonary congestion
D
Disability
• Fever/gastroparesis
• Raised procalcitonin
• Sepsis
• Skin Eschar cultures
• Narrow spectrum antibiotics
Wound care
• Rehab/Reconstructive surgery
• Re-integration
Subsequent management

(CRT: capillary refill time; DVT: deep vein thrombosis; ECHO: echocardiogram; ENT: ear, nose, and throat; ET: endotracheal; GEDV: global end-diastolic volume; ICU: intensive care unit; IVC: inferior vena cava; MAP: mean arterial pressure; NG: nasogastric; UO: urine output)

Source: Flowchart created by I Menon.

SUMMARY

Caring for the critically ill burns patient is a challenge for ICUs. Optimal initial evaluation, monitoring, and management are vital to prevent or at least limit the multiorgan damage that accompanies these injuries. It is recommended that the intensivist avoids excessive fluid administration, prophylactic antibiotics, and unnecessary ET intubation. Frequent monitoring of the airway with the help of video laryngoscopy and tracheal ultrasound is advised with expert intubation when needed. Early and repeated debridement of devitalized tissue is done and escharotomies or fasciotomies are performed where required. These procedures could be accompanied by excessive blood loss requiring transfusion of blood products on occasion. Sepsis and renal failure are the major complications and outcome determinants of critically ill burns patients. Early rehabilitation and attention to nutrition facilitates recovery **(Flowchart 2)**.

Regular counseling services for the patient and the relatives is essential.

In fact, the outcome of a critically ill burns patient admitted to a critical care unit could be a reflection of the quality and safety standards of the ICU.

ACKNOWLEDGMENT

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Chapter 20

How will I Examine a Patient with Neuromuscular Weakness?

Pralay Shankar Ghosh, Banani Poddar

ABSTRACT

Neuromuscular weakness is not an uncommon problem in the intensive care unit (ICU). One may present with such a problem while others may develop this during an ICU stay. The diagnosis is challenging as proper medical history and clinical examination often take backstage while resuscitation becomes the primary objective. It is important to perform a systematic, albeit abridged clinical examination of such a patient. A basic understanding of differential diagnosis and a protocolized approach often help in better patient management.

INTRODUCTION

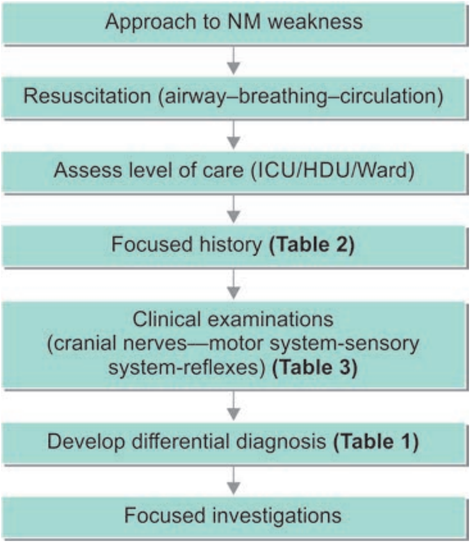
Neuromuscular weakness is often encountered in critically ill patients. In the critically ill, generalized neuromuscular weakness is due to one of the following:

- An acute new-onset neurological disorder, e.g., Guillain–Barré (GB) syndrome
- An acute deterioration in a preexisting neurological disease, e.g., myasthenia gravis with myasthenic crisis
- A new-onset weakness in a critically ill patient with a non-neurological illness; e.g., critical illness polyneuropathy (CIPN).

In the first two conditions, the patient would present to the intensive care unit (ICU) with neuromuscular weakness, which may either progress or be at its peak at presentation and improve with therapy, depending on how early in the course of the disease the patient is admitted. In the last condition, a patient who is already in the ICU for some other illness is found to have neuromuscular weakness when the underlying disease is improving, and the ICU team is contemplating weaning from mechanical ventilation.

Neuromuscular weakness predisposes these patients to compromise of airway and breathing when the muscles responsible for maintaining these two vital functions are affected. Hence, it is very important to quickly identify such emergencies and stabilize the airway and breathing before completing the evaluation of the patient **(Flowchart 1)**.

Flowchart 1: Approach to neuromuscular (NM) weakness.


Approach to NM weakness
Resuscitation (airway–breathing–circulation)
Assess level of care (ICU/HDU/Ward)
Focused history **(Table 2)**
Clinical examinations (cranial nerves—motor system-sensory system-reflexes) **(Table 3)**
Develop differential diagnosis **(Table 1)**
Focused investigations

This is followed by focused history, clinical examination, and a few laboratory investigations and/or electrophysiological tests to make a diagnosis. A detailed neurological examination is not possible in this situation due to the patient's inability to perform many of the tests, the presence of an endotracheal tube (ETT), and various catheters/restraints, and the patient may be under the effect of sedation.

In this chapter, we will discuss how an intensivist examines a patient with neuromuscular weakness and the various investigations that help in arriving at a diagnosis.

CASE SCENARIOS

1. *A previously healthy 18-year-old male is brought to the ICU with acute generalized weakness. 2 days ago, he noticed some difficulty in walking which progressed to inability to stand without support by the evening. Over the next day, he noticed weakness of the fingers and gradually this progressed to affect the entire upper limbs. Since the morning of the admission day, he found it difficult to swallow and had drooling, followed by difficulty in breathing. On admission, he is conscious but looks anxious and he is sweating; pulse rate is 100 beats/min, respiratory rate is 40 breaths/min with jerky breathing and poor chest rise, blood pressure on noninvasive measurement is 130/80 mm Hg, and pulse oximetry shows an oxygen saturation of 89% with 6 L/min oxygen via face mask. Pooling of oral secretions with drooling is seen. Flaccid paralysis is noted with symmetrical involvement of all four limbs and areflexia. Arterial blood gas (ABG) shows respiratory acidosis. A decision is taken to perform an endotracheal intubation and connect him to a ventilator.*
2. *A 65-year-old gentleman, a known patient of chronic obstructive pulmonary disease (COPD) and diabetes mellitus, was admitted with community-acquired pneumonia with septic shock. He had two previous hospital admissions for exacerbation of COPD. During this admission, he required mechanical ventilation for over 10 days, and he was treated with broad-spectrum antibiotics, namely meropenem and polymyxin B based on cultures. During the first 24 hours of ventilation, he was paralyzed with a neuromuscular blocking agent in view of ventilator asynchrony. He gradually improved and vasopressors were weaned off and ventilatory support was weaned. On day 12, since he was hemodynamically stable and required fraction of inspired oxygen (FiO_2) of 0.3, the treating intensivist contemplated a spontaneous breathing trial. After a sedation vacation of 24 hours, the patient was able to open his eyes and follow simple verbal commands but was found to have generalized weakness making weaning/extubation difficult at that moment. The weakness involved all four limbs and was bilaterally symmetrical in nature. The intensivist noted generalized wasting of limb muscles, hypotonia of all four limbs, and diminished deep tendon reflexes.*

CAUSES OF NEUROMUSCULAR WEAKNESS

Neuromuscular weakness may result due to involvement of the central nervous system (CNS), spinal cord, or peripheral nervous system (PNS). In the PNS, this can be further defined as involvement of different parts of a motor unit, namely anterior horn cell, peripheral nerve, neuromuscular junction, and muscle.

Weakness due to CNS lesions such as stroke is asymmetric and is often associated with altered sensorium and cranial nerve palsies. Brain stem lesions, on the other hand, often present with generalized weakness. These include vascular conditions (e.g., brainstem infarct or hemorrhage), brainstem encephalitis, and osmotic demyelination syndromes. Some of these may result in a "locked-in state."

Table 1 lists the various illnesses which affect the spinal cord and the PNS at different levels and give rise to neuromuscular weakness.

INITIAL ASSESSMENT

Initial assessment is targeted to stabilization of airway, breathing, and circulation. Severe weakness associated with poor sensorium or poor airway reflexes as a result of cranial nerve involvement may demand for airway protection leading to tracheal intubation. Ventilatory support in the form of noninvasive ventilation (NIV) may be required in a conscious patient with weakness of the respiratory muscles.

The clinical examination tools for airway and respiratory weakness:

- *Airway assessment:* Inability to lift the head from the bed suggests weakness of the central muscles and is usually associated with weakness of the muscles which maintain a clear airway. Drooling and ineffective cough represent inability to protect the airway.
- *Respiratory muscle weakness:* Increased respiratory rate with shallow breathing, orthopnea, and use of accessory muscles may be due to weakness of the diaphragm. Diaphragmatic weakness could also result in an abdominothoracic pattern of breathing.

A single-breath count may help in the assessment of respiratory reserve. Studies in patients with myasthenia gravis suggest that counts <20–25 indicate abnormal respiratory function, whereas values <7 are considered abnormal in patients with GB syndrome. Absence

TABLE 1: Diseases presenting with generalized neuromuscular weakness, classified by localization.

Muscle diseases	***Neuromuscular junction disorders***	***Peripheral neuropathies***	***Anterior horn cell disorders***	***Spinal cord disorders***
• Critical illness myopathy • Hypokalemic myopathy • Hypophosphatemia • Inflammatory myopathies: Polymyositis, dermatomyositis • Rhabdomyolysis • Muscular dystrophies *Myotonic dystrophy* *Mitochondrial myopathies* *Acid maltase deficiency*	• Organophosphorus poisoning • Myasthenia gravis • Snake bite • Neuromuscular blocking agent-induced weakness • Botulism • Antibiotic-induced myasthenia *Insect/marine toxins* *Lambert–Eaton myasthenic syndrome* *Congenital myasthenic syndromes* *Hypermagnesemia* *Tick paralysis*	• Critical illness polyneuropathy • Guillain–Barré syndrome [acute inflammatory demyelinating polyneuropathy (AIDP)] • Porphyric neuropathy • Chronic idiopathic demyelinating polyneuropathy (CIDP) *Phrenic neuropathies* *Toxic neuropathy* *Vasculitic neuropathy* *Diphtheria* *Lymphoma* *Cytomegalovirus-related polyradiculoneuropathy*	• Amyotrophic lateral sclerosis • Paraneoplastic motor neuron disease • West Nile virus infection • Spinal muscular atrophy • Acute poliomyelitis	• Trauma • Hematoma • Epidural abscess • Demyelination: – Acute disseminated encephalomyelitis, transverse myelitis, neuromyelitis optica, multiple sclerosis – Spinal cord infarction *Infective myelitis: Coxsackievirus A, B, cytomegalovirus, Mycoplasma, Legionella, Herpes* *Paralytic rabies ("dumb rabies")*

Note: Conditions given in *italics* are less common

of an audible cough is an abnormal finding when a patient is asked to cough forcefully.

Pulse oximetry and ABG are useful bedside tools. ABG may be normal initially until respiratory muscles become weak enough to cause hypercarbia. Static maximal inspiratory pressure maneuver and sniff nasal inspiratory pressure are among the few proposed tests to evaluate respiratory pump failure.

Baseline assessment and serial monitoring of clinical parameters and investigations are needed to identify gradual deterioration.

There are many disease-specific indicators that can guide ICU admission and the need for respiratory support. For example, rapid progression of weakness, evolving respiratory distress, severe dysphagia, or dysautonomia and EGRIS score >4 guide well as indicators of ICU admission in GB syndrome [EGRIS (Erasmus GBS Respiratory Insufficiency Score)].

FOCUSED HISTORY

The two cases mentioned in the beginning require two different approaches. However, in both cases we need to take a focused history to rule out preexisting neurological illness, to rule out relevant risk factors and to understand the evolution of the illness which may guide us in the diagnosis.

A pattern of questions that may guide us is shown in **Table 2**.

CLINICAL EXAMINATION AND INVESTIGATIONS

General Examination

Observe the general build of the patient and the appearance of anxiety or distress.

Skin rashes suggestive of vasculitis or typical skin changes of dermatomyositis (heliotrope rash and Gottron papules) could give a clue to the diagnosis. Look carefully for fang marks over the distal limbs, if the history is suggestive of neuroparalytic snake envenomation (e.g., krait bite). Local necrosis and limb swelling are characteristic of cobra bite.

The examination of the airway and respiratory system takes priority in all cases suspected to have neuromuscular weakness, as already mentioned earlier. Airway compromise may be life-threatening and an advanced airway should be secured first.

TABLE 2: Focused history in a patient with neuromuscular weakness and its interpretation.

Focused history to be taken	***Implication***
Any infective event prior to this illness: Diarrhea or upper respiratory infection	Guillain–Barré syndrome
History of vaccination prior to the illness	Guillain–Barré syndrome or transverse myelitis
Onset: Acute or chronic	To differentiate acute illnesses (Guillain–Barré syndrome) from chronic illnesses [e.g., chronic inflammatory demyelinating polyneuropathy (CIDP)]
High-grade fever with chills and rigors at onset of illness	Infectious causes such as epidural abscess or pyomyositis
Pattern of progression: Ascending, descending, or no specific pattern	Guillain–Barré syndrome is typically an ascending weakness, whereas botulism is typically a descending weakness
Worsening of symptoms as the day progresses or seen only after exertion	Myasthenia gravis
Presence of ptosis	Myasthenia gravis, neurotoxic snake envenomation
Any tingling/paresthesias or band-like sensations: Either at onset or subsequently	Typically seen with transverse myelitis
Painful/tender muscle involvement	To rule out polymyositis
Bladder/bowel involvement	Spinal cord lesions, especially transverse myelitis
History of drug intake during current illness or previously	• Several drugs, especially in the critical care set up can predispose to neuromuscular weakness, e.g., aminoglycosides • Neuromuscular blockers, steroids, statins, antiretroviral agents, and several antibiotics can lead to generalized weakness.
History of trauma (head or spinal injury)	Traumatic spinal cord injury
History of chronic critical illness	Suggests critical illness neuropathy or myopathy
Environmental history	To find circumstantial evidence to implicate snake venom poisoning
Similar history in any other family member	Porphyria, periodic paralysis, etc. may be present in other family members
History of episodic abdominal pain, skin lesions, and waxing and waning course. May be associated with red-colored urine during an episode of abdominal pain	Porphyria
History of unaccustomed prolonged exercise, especially in very hot weather. Associated history of passage of cola-colored urine	Rhabdomyolysis
Past history of similar weakness which was transient	To rule out any form of periodic paralysis
History of autoimmune diseases	Transverse myelitis
Recurrent episodes of similar weakness, some prolonged and requiring respiratory support	Chronic inflammatory demyelinating polyneuropathy
History of drooling or postprandial cough	Suggests bulbar weakness
Difficulty in breathing	To understand the extent of involvement as respiratory muscle involvement is seen last
The associated loss of consciousness	• Loss of consciousness is a late feature of neuromuscular weakness suggesting hypoxemia/hypercarbia • Loss of consciousness at onset is suggestive of a cortical insult

Examination of the Nervous System

An appropriate order of examination is the assessment of sensorium or cognitive status, cranial nerves, speech, motor system, and sensory system.

Assess Sensorium/Higher Function

- *Conscious patient:*
    - Ask directed questions to assess orientation, attention
    - Assess for delirium (use standard delirium scores, e.g. Confusion assessment method for the ICU or CAM-ICU)
    - Check for Glasgow Coma Scale (GCS)
- *Unconscious patient:*
    - Check for response, i.e., alert, response to voice or pain or no response at all (AVPU) and purposeful movement (indicating intact higher function)
    - Check GCS
- *Exclude seizure:* Limb movement, up rolling of eyes, and postictal confusion.

Cranial Nerve Examination

All cranial nerves can be tested in an awake and alert patient who can cooperate while only a few of the cranial nerves can be tested in unconscious patients.

A thorough evaluation of the different cranial nerves is not possible in a situation as described in the case scenarios. The following can be tested quickly in conscious patients.

- *Cranial nerve 1:* This nerve is not tested.
- *Cranial nerves 2, 3, 4, and 6:* These can be tested by checking the pupillary response to light and movements of the eyes (both in horizontal and vertical directions)
- *Cranial nerve 5:* The presence of conjunctival and corneal reflexes and sensations over the face are checked to test this cranial nerve.
- *Cranial nerve 7:* Palsy of bilateral seventh cranial nerves leads to mask-like facies, with the inability to completely close the eyelids. In a patient with no such obvious palsy, testing can be done by asking the patient to firmly close their eyes and to blow out his cheeks and checking the strength of each of these.

Unilateral palsy (frequently seen in stroke patients) causes deviation of the angle of the mouth to the side opposite to the side of the lesion.

- *Cranial nerve 8:* This nerve is not tested.
- *Cranial nerves 9 and 10:* This is difficult to test in patients with ETT in situ. Drooling is a sign of palsy of these cranial nerves, leading to pharyngeal weakness.

The presence of a gag reflex and coughing/bucking on movement or suctioning of the ETT suggest 9th and 10th cranial nerve function.

Note: It is mandatory to do a proper evaluation of swallowing once a patient with neuromuscular weakness is extubated or is tracheostomized and oral feeding is anticipated.

- *Cranial nerve 11:* This can be tested by assessing shrugging of the shoulders.
- *Cranial nerve 12:* Movements of the tongue are checked to test the 12th cranial nerve.

In an unconscious patient, pupillary size, symmetry, and response to light is all that can be ascertained along with conjunctival and corneal reflexes. The presence of "doll's eye movements" on passive side-to-side head movement indicates an intact brain stem function with 3rd, 4th, 6th, and 8th cranial nerves' function.

Assess Motor Function (Bilaterally)

- Assess for bulk and tone
- Observe for active and passive movement
- Check movement against resistance bilaterally
- If unable to follow commands, check response to pain
- Try to differentiate proximal versus distal weakness
- Grade the power of the muscles at all joints using the Medical Research Council (MRC) grading. Power of the muscles is graded from 0 to 5, where grade 0 represents no movement at all and grade 5 is normal movement against active and full resistance. A composite score of all the muscle groups is then obtained. A score <48/60 implies significant weakness (especially when used for assessment of ICU-acquired weakness).

If asymmetrical weakness, consider if the patient has a CNS lesion and differentiate between upper motor neuron (UMN) type of weakness as compared to lower motor neuron (LMN) type of weakness **(Table 3)**.

Assess Sensory Function (Bilaterally)

- Check for pain and temperature
- Light touch/pressure
- Vibration and proprioception are usually not tested as it is difficult to obtain cooperation of the patient for this.

TABLE 3: Clinical differentiation of upper motor neuron pattern of weakness from lower motor neuron pattern of weakness.

Upper motor neuron lesion	***Lower motor neuron lesion***
Increased tone	Flaccid paralysis
Extensor plantar response	Mute plantar reflex or normal response
Exaggerated deep tendon reflexes	Decreased deep tendon reflexes
Clonus	Fasciculations

Deep Tendon Reflexes

Elicit the following reflexes as demonstrated in **Figures 1 to 5**.

- Biceps brachii (C5, C6)
- Triceps (C7, C6)
- Brachioradialis (C5, C6)
- Quadriceps (L2, L3, L4) (also called knee jerk)
- Achilles (S1) (also called ankle jerk).

Cerebellar Function

Cerebellar function is usually not assessed in a patient with neuromuscular weakness.

Autonomic Dysfunction

Autonomic dysfunction is associated with some disorders. This is mostly represented by tachycardia and fluctuating blood pressure.

Specific Investigations

Laboratory investigations: Serum creatine kinase may be elevated in primary muscle disease. Serum electrolytes should be evaluated to rule out hypokalemic paralysis, hypophosphatemia, or hypermagnesemia. Evaluation of the cerebrospinal fluid (CSF) for albuminocytological dissociation (raised protein but normal cell count) is indicated when GB syndrome is suspected.

The ABG would show hypercarbia early in the course of the disease. Hypoxemia is a late feature and is usually seen in patients with impending respiratory arrest.

Further investigations which are required for a definitive diagnosis (not bedside tests) include:

- *Electrophysiological studies:* Electromyography (EMG) and nerve conduction velocity (NCV) studies
- Muscle or nerve biopsy
- Imaging to rule out spinal cord lesions or CNS lesions.

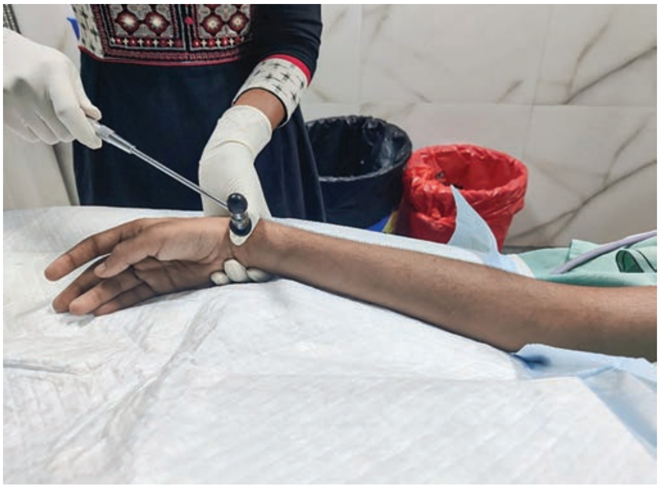


Fig. 1: Brachioradialis jerk.

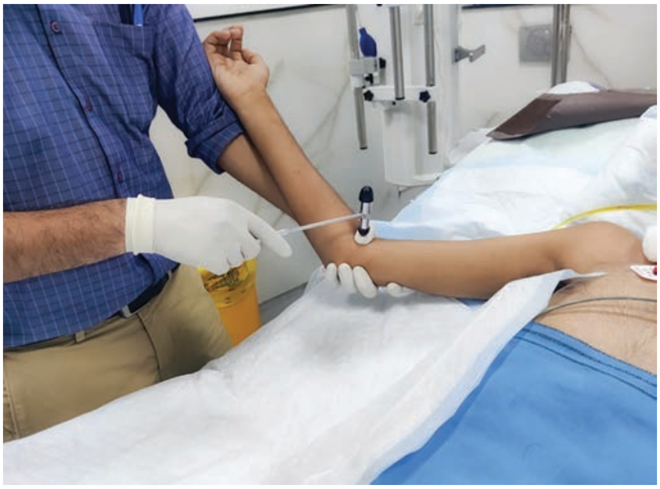


Fig. 2: Biceps jerk.

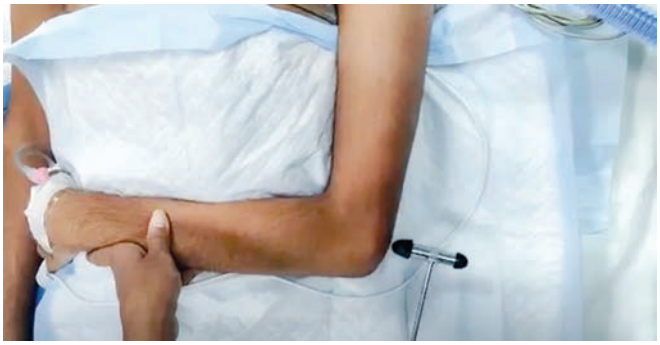


Fig. 3: Triceps jerk.

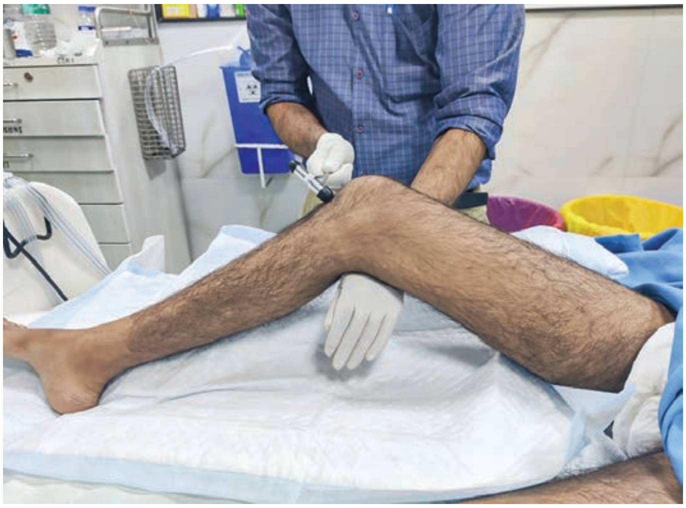


Fig. 4: Knee jerk.

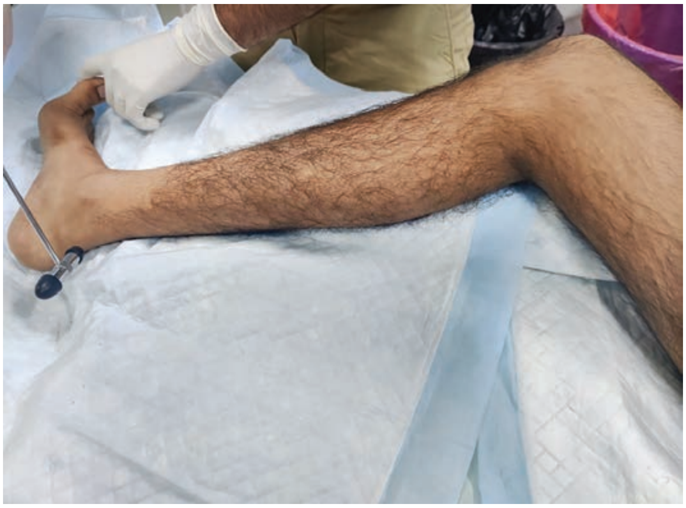


Fig. 5: Ankle jerk.

CHRONIC PRESENTATION

In patients who are in the ICU for prolonged periods (as in case scenario 2), with certain risk factors including use of neuromuscular blocking agents, hyperglycemia, sepsis and acute kidney injury, a diagnosis of CIPN, critical illness myopathy, or a combination of the two is considered **(Table 4)**. The following features are seen in these conditions.

Table 5 gives an overview of the common conditions of the PNS, which are associated with neuromuscular weakness in ICU patients.

TABLE 4: Important clinical features of critical illness neuropathy and myopathy.

Critical illness polyneuropathy (CIPN)	***Critical illness myopathy (CIM)***
Sensorimotor polyneuropathy: • Limb muscle weakness and atrophy • Reduced or absent deep tendon reflexes • Loss of peripheral sensation to light touch and pinprick • Relative preservation of cranial nerve function	*Myopathy:* • Flaccid quadriparesis that may affect proximal more than distal muscles • Failure to wean from mechanical ventilation

TABLE 5: An overview of clinical features of the most common causes of neuromuscular weakness at different levels of the PNS.

Anatomical level	*Common diseases*	*Weakness seen in*	*Symmetry of weakness*	*Proximal vs. Distal*	*UMN/LMN type of weakness*	*Tone*	*Tendon reflexes*	*Atrophy seen or not*	*Other signs noted during examination*	*Sensory loss*	*Any other notable feature*
Spinal cord	Trauma	Torso, limbs	Bilateral	Both	LMN type initially, in long term UMN type	Flaccid initially, later hypertonia	Decreased initially, later increased	Yes	Different patterns of weakness/ "syndromes"	Definite sensory level	Autonomic dysfunction if involvement above T6
Anterior horn cells	Amyotrophic lateral sclerosis	Limb, bulbar, axial, and respiratory	Asymmetrical	Distal	UMN or LMN	Flaccid in LMN	Decreased in LMN	Yes in LMN	Fasciculations in LMN	Sometimes	Autonomic dysfunction, cranial nerve involvement
Neuropathy	Guillain–Barré syndrome	Limb, bulbar, axial, and respiratory	Bilateral	Both	LMN	Flaccid	Reduced or absent	Yes	None	Tingling paresthesia	Autonomic dysfunction, cranial nerve involvement
	Critical illness polyneuropathy	Limbs affected more than others	Bilateral	Both	LMN	Flaccid	Reduced or absent	Marked	None	Often	Other features of undernutrition present
Neuromuscular junction	Myasthenia gravis	Eye, face, neck, limb, and respiratory	Unilateral or bilateral	Proximal more than distal	LMN	Flaccid	Decreased	No	None	No	Mainly ocular, fluctuating weakness
Myopathy	Critical illness myopathy	Limbs, torso	Bilateral	Proximal more than distal	LMN	Reduced	Reduced	Yes	None	No	Leads to weaning difficulty

(LMN: lower motor neuron; PNS: peripheral nervous system; UMN: upper motor neuron)

SUMMARY

To summarize, neuromuscular weakness is encountered in a sizeable number of critically ill patients. A detailed neurological assessment in such a patient is often limited by artificial airway and sedation and/or a need for resuscitation. However, a systematic albeit limited clinical examination coupled with knowledge of anatomy hold the key to a successful approach to such a patient.

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Chapter

21

How will I Examine a Patient with Gastrointestinal Bleeding?

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ABSTRACT

Gastrointestinal bleeding (GI bleeding) has become an uncommon reason for index intensive care unit (ICU) admission (in particular those who are hemodynamically unstable). In patients who seek medical attention for GI bleeding, need for surgery and procedures have significantly reduced over the years, through early recognition, aggressive reflux disease management, and likely lifestyle changes. Also, critically-ill patients (who are admitted for nonbleeding causes), have been developing various forms of GI bleeding during their ICU stay (predominantly stress ulcer bleeding). Often, the GI bleeding is a common medical emergency needing intensive care and requires aggressive resuscitation when it is massive and associated with hypotension. Whilst upper GI bleeding predominate ICU admissions, occasionally sicker patients with lower GI bleeding makes for a challenging scenario. It is imperative to stabilize the patient, control bleeding quickly, find the cause of bleeding, and identify the underlying disease.

Understanding anatomical boundaries to differentiate upper, middle versus lower bleeding site is just a beginning and this should further involve likely sources that predispose to severity of illness necessitating ICU admission. Knowing the predisposing causes such as liver cirrhosis, prior anticoagulant use, prior peptic ulcer disease, etc., might make assessment and management more accurate.

This requires a rapid and focused examination of the patient. The critical care physician should elicit appropriate history, and specific clinical signs during evaluation. A focused assessment and in return an appropriate management plan may result in improved outcome. This chapter focuses on how to examine the patients who present with GI bleeding, provides a systematic approach to the examination of such patients, and specific clinical signs to elicit.

INTRODUCTION

Gastrointestinal bleeding (GI bleeding) as an index diagnosis that needs intensive care unit (ICU) admission is usually, an acute or acute on chronic phenomenon. Based on the frequency of presentation apart from differing causes, upper and lower GI bleeding are discussed separately in this section for ease of understanding. The ligament of Treitz anatomically differentiates upper from lower GI bleeding based on the source. Understanding anatomical boundaries to differentiate upper versus lower bleeding sites is just the beginning **(Fig. 1)**. Apart from anatomical differentiation, specific causes of GI bleeding that predispose to illness severity necessitating ICU admission are to be considered during evaluation.

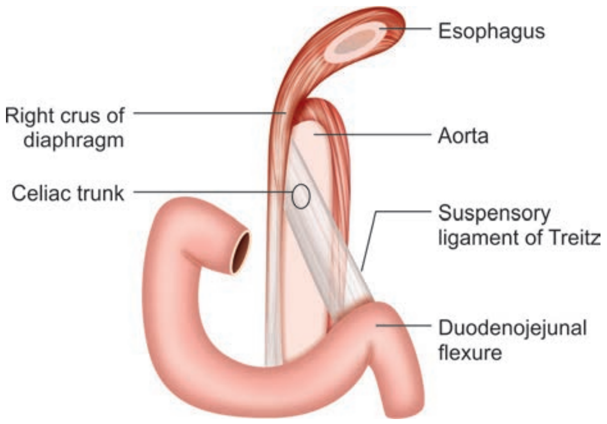

Fig. 1: Anatomical boundary upper versus lower gastrointestinal bleeding site.

Upper gastrointestinal bleeding (UGIB) tends to be more severe and has diverse reasons compared to lower gastrointestinal bleeding (LGIB) based on pathology and is usually associated with a higher need for resuscitation, transfusion of blood products, and corrective procedures. LGIB (with or without hemodynamic instability) is an uncommon reason for ICU admission.

In view of a low admission frequency and different clinical etiologies compared to ward-level patients, a structured assessment and management will result in appropriate care and in turn outcome. Knowing the predisposing causes such as anticoagulant use, liver cirrhosis, and prior bleeding episodes might make assessment and management more accurate. Assessment of this group of patients should initially involve shock severity and later general and specific examinations for the cause and site of bleeding. Increasingly point-of-care ultrasound (POCUS) is being utilized for both shock assessment and monitoring of resuscitation apart from its use to diagnose underlying conditions.

UPPER GASTROINTESTINAL BLEEDING SCENARIOS

Case Scenarios

1. *A 67-year-old female presented with comorbidities such as ischemic heart disease (IHD) on dual antiplatelets, chronic kidney disease (CKD) stage 2, diabetes, and hypertension (HTN).* History *includes gangrenous pyelonephritis leading to septic shock and acute kidney injury (AKI) requiring mechanical ventilation, dialysis, vasopressor support, and stress dose steroids. The patient has been ill and had nasogastric (NG) aspiration of altered colored blood along with melena with significant hemodynamic instability.* Initial evaluation *determined cold and clammy, heart rate (HR) 135 beats with 80 systolic blood pressure (SBP), and arterial blood gas (ABG) showing hemoglobin (Hb) of 5 g/dL, and lactate 5.4;* Impression: *Likely stress ulcer-related bleeding.*
2. *A 54-year-old male presented with comorbidities such as alcoholic liver disease and HTN.* History *includes recent admission for worsening jaundice, 4 days after discharge started, passing melena and red blood in stools, and moderate to large quantity of blood in vomit on the day of evaluation.* Initial evaluation *determined warm peripheries but capillary refill 3–4 seconds; deeply jaundiced, HR 124, SBP 92, ABG showing Hb of 6 gm/dL and lactate of 9;* Impression: *Likely variceal upper GI bleeding (UGIB).*
3. *A 24-year-old male presented with no comorbidities.* History *includes that after attending a wedding presents with multiple episodes of vomiting and retching followed by small to moderate amount of blood in vomit.* Initial evaluation *determined HR 135 with SBP of 110, and ABG showing Hb of 14 g/dL, and lactate 2.1.* Impression: *Likely Mallory–Weiss syndrome.*
4. *A 74-year-old male presented with comorbidities such as IHD and peptic ulcer disease.* History *includes multiple days of melena and fatigue; seen in gastroenterology outpatient department (OPD) and was evaluated for clinical anemia and blood loss with upper GI endoscopy. Evaluation notes Dieulafoy's lesion.* Initial evaluation *determined warm peripheries but capillary refill 3–4 seconds, deeply jaundiced, HR 101, SBP 164, ABG showing Hb of 7.2 g/dL, and lactate of 2;* Impression: *Monitoring for rebleeding of Dieulafoy's lesion.*

History and Symptomatology—Upper Gastrointestinal Bleeding

To elicit a focused history, one needs to be aware of the etiology that is likely to lead to an ICU admission. The above scenarios point us to varied presentations that are different from outpatient or ward-care groups from which the etiological frequency is derived.

The UGIB in ICU is divided grossly as one where bleeding is primarily related to the index admission or due to a cause that directly caused GI bleeding leading to the admission. Over the years, peptic ulcer-related severe UGIB has reduced significantly but the incidence of variceal UGIB remains stable. In recent years, diagnoses that were deemed less common previously form significant reasons for ICU admission like Mallory–Weiss syndrome, malignancy-related ulcers, angiodysplasia, and esophagitis **(Table 1)**. A quick and focused history-taking will be useful in acute UGIB emergencies **(Table 2)**.

LOWER GASTROINTESTINAL BLEEDING

Introduction

Evaluation of critically-ill patients presenting with LGIB is consistent with general principles of examination but the focus should be on etiological differences noted with

TABLE 1: Specific upper gastrointestinal bleeding causes for an intensive care unit admission.

Peptic ulcer disease	Critical illness-related stress gastropathy
Esophageal and gastric portal hypertensive changes	Mallory–Weiss syndrome
Esophagitis	Malignancy-related bleeding
Postprocedure monitoring (variceal banding/postulcer bleeding treatment)	Monitoring for Dieulafoy's lesion

TABLE 2: History relevant to upper gastrointestinal bleeding needing an intensive care unit admission.

Shock or impending shock symptoms	
CVS	Postural hypotension or hypotension, tachycardia, palpitations, etc.
CNS	Falls, loss of conscious state, or altered sensorium
Symptoms relevant to severity of bleeding	
Duration	• Acute versus acute on chronic • *Acute:* <3 days of blood loss • Acute is more likely to be associated with hemodynamic/organ dysfunction
Frequency	Higher with variceal compared to other
Quality	• Fresh Blood in vomit (variceal > ulcer bleed) • Altered colored blood in vomit (ICU bleeding > Mallory–Weiss; could be upper respiratory bleed), when blood stays long in stomach it turns color due to digestion by partly gastric acid. • Associated melena (>24 hours of bleeding onset) • Hematochezia (higher volume of loss or small bowel disease)
Quantity	• Usually moderate to severe (quantification judged not based on quantity of blood but the effect it has on the body, e.g., Hb level, blood pressure, etc.) • Need for blood transfusion prior to ICU admission
Associated symptoms relevant to underlying pathology	
Painless excess blood loss	Variceal bleeding
Painful excess blood loss	Peptic ulcer disease
Painful mild to moderate blood loss	Mallory–Weiss syndrome/esophagitis
Abdominal distension	Portal hypertension related
Past history	Peptic ulcer disease, portal hypertension, ICU admission severity, malignancy, angiodysplasia
Recent procedures	Endo/colonoscopy, contrast scans of intestine, and abdominal surgery
Medications	Anticoagulants, aspirin/NSAIDs
Immunosuppresssion (disease/medications)	Rarer causes such as esophageal candidiasis, CMV, and lymphomatous disease

(CVS: cardiovascular system; CMV: cytomegalovirus; CNS: central nervous system ICU: intensive care unit; Hb: hemoglobin; NSAIDs: non-steroidal anti-inflammatory drugs)

ICU admission, presentation mode, and associated or impending organ dysfunction. Below is a structured approach to guide one to evaluate an LGIB case but it is not an absolute guide by itself.

The LGIB refers to a source of blood loss which is distal to the ligament of Treitz. Though LGIB is referred predominantly as one arising from the colon and the anorectum, bleeding from small bowel or mid-colonic area (between the ligament of Treitz and the ileocecal valve) is now considered to be middle gastrointestinal bleeding.

ICU admission or triage for the same is rarely due to the quantity of blood loss but acuity of loss and consequent physiological stress on individuals with significant comorbidities.

Classically, acute LGIB originating from either the colon or the rectum, manifests as hematochezia (maroon or red blood passed through the rectum). Uncommonly, LGIB can manifest as melena (black, tarry stools), or, conversely, brisk (rapid) UGIB can manifest as hematochezia.

Case Scenarios

1. *An 84-year-old male presented with comorbidities such as IHD with low ejection fraction on dual antiplatelets and β-blockers, CKD stage 2, diabetes, and HTN.* History *includes passing red stools multiple times during the day brought to emergency room (ER) following collapse at home.* Initial evaluation *determined cold and clammy, HR 68 with 80 SBP, and ABG showing Hb of 5 g/dL, and lactate 2.4.*
2. *A 48-year-old male presented with comorbidities diabetes mellitus (DM)/HTN.* History *includes recent surgery for ileocecal region malignancy with anastomosis; On day 4 of illness, passing melena and red blood in stools; medical emergency team (MET)/rapid response team (RRT) raised due to severe anemia, fever, tachycardia, and looking unwell; Deemed anastomotic site bleeding.* Initial evaluation *determined warm peripheries but capillary refill 3–4 seconds; HR 124, SBP 92, and temperature 102.2°F; ABG showing Hb of 6 g/dL and lactate of 5.6.*
3. *A 42-year-old male presented with a family history of colon cancer.* History *includes underwent elective colonoscopy; multiple polyps-suspicious of malignancy; polypectomy performed; bleeding risk deemed high; and admission to ICU for monitoring.* Initial evaluation determined *HR 92 with 130 SBP, and admission ABG showing Hb of 14 g/dL with lactate of 0.8.*

History and Symptomatology—Lower Gastrointestinal Bleeding

To elicit a focused history, one needs to be aware of the etiology that is likely to lead to an ICU admission. *See* **Table 3** for focused history to take in cases of LGIB. Literature is sparse for such specific LGIB causes. The above scenarios point us to varied presentations which are different to outpatient or ward-care group from which the etiological frequency is derived from.

TABLE 3: History relevant to intensive care unit in cases of lower gastrointestinal bleeding.

Shock or impending shock symptoms	
CVS	Postural hypotension or hypotension, tachycardia, palpitations, etc.
CNS	Falls, loss of conscious state, or altered sensorium
Symptoms relevant to severity of bleeding	
Duration	• Acute versus acute on chronic • *Acute:* <3 days of blood loss • Acute is more likely to be associated with hemodynamic/organ dysfunction
Frequency	Higher with inflammatory or infective colitis
Quality	Passing of blood mixed with stool or not
Quantity	Hematochezia or not
History relevant to underlying pathology	
Painless excess blood loss	• Diverticular disease • Hemorrhoids
Painless mild-moderate blood loss	Polyps, malignancy, and ischemic colitis
Painful mild-moderate blood loss	Inflammatory bowel disease, infective bowel disease
Painful excess blood loss	Inflammatory bowel disease
Abdominal distension	Obstruction due to malignancy, peritonitis from diverticular disease, etc.
Hematemesis	Indicates an upper gastrointestinal source or mixed pathology
Weight loss	Suggestion of chronic illness including malignancy
Past history	Diverticulosis, inflammatory bowel diseases, polyps, malignancy, tuberculosis, angiodysplasia, and severe atherosclerotic disease predisposing ischemia
Recent procedures	Endo/colonoscopy, contrast scans of intestine, and abdominal surgery
Medications	Anticoagulants, aspirin/NSAIDs
Immunosuppresssion (disease/medications)	Rarer causes such as *C. difficile*, CMV, and lymphomatous disease

(*C. difficile*: *Clostridium difficile*; CMV: cytomegalovirus; CNS; central nervous system; CVS; cardiovascular system; NSAIDs: nonsteroidal anti-inflammatory drugs)

Diverticular disease, inflammatory bowel diseases, and tumors (polyps and malignancy) account for majority of the LGIB in western literature (not specific to ICU). In comparison, Indian literature notes tumors, inflammatory bowel diseases, and enteric ulcers (nonspecific and

TABLE 4: Specific lower gastrointestinal bleeding causes for an intensive care unit admission.

Colorectal malignancy including polyps (include postprocedure state)	*Angiodysplasia:* Predominantly in elderly and radiation induced
Inflammatory bowel diseases: Predominantly ulcerative colitis	Ischemic colitis
Small bowel ulcers (enteric/tubercular or other)	Hemorrhoids (includes portal hypertensive process)

specific) as the main causes again not specific to ICU. **Table 4** gives an abbreviated list pertinent to ICU.

EXAMINATION OF A PATIENT WITH GASTROINTESTINAL BLEEDING

One could start an objective assessment with an airway, breathing, circulation (ABC) approach and/or consider specific features that might confirm or give a clue to the severity of bleeding as well as the cause.

The *ABC approach* is designed to quickly assess and treat life-threatening bleeding or associated organ dysfunction.

- *Conscious state:* Hypotension contributing to low Glasgow Coma Score (GCS). Clue to severity of bleeding.
- *Airway and breathing:* Can the patient protect the airway? It depends upon the mental status, quantity of bleeding (Upper GI), and the risk of recurrent bouts of UGIB (risk of aspiration in GI bleeding). Watch for reduced (encephalopathy-related) or acidotic breathing patterns and monitor oxygen saturation (SpO_2).
- *Circulation:* Blood pressure, heart rate, capillary refill time, and clammy skin are indicators of severity of hemorrhage. Remember stages of hemorrhagic shock.

Specific Examination for Assessment of Shock

- Low SBP/tachycardia/(Shock index = *HR/SBP:* <1/>1)
- Lactate levels (>2 mmol/L)
- *Dynamic over static indices of preload:* Inferior vena cava (IVC) collapsibility/distensibility index.
- Passive leg raise (PLR)/fluid challenge (250–300 mL) in conjunction with pulse pressure variation (PPV) or velocity time integral (VTi) variation/IVC indices.
- Extended-focused assessment sonography in trauma (E-FAST), if traumatic cause is suspected.

Alternate Pathway to General and Focused Assessment of a Patient with Gastrointestinal Bleeding

General Physical Examination

Concurrent measurement of vitals (HR, respiratory rate, and blood pressure) along with examinations for signs of anemia severity, examination of extremities, mental state, urine output, peripheral edema (indicate an acute on chronic pathology), lymph nodes (more likely with upper GI pathology), or other features consistent with chronic liver disease are essential. If active bleeding is suspected or found, the above findings should indicate hemorrhagic shock class (I–IV) and in turn possible quantity of loss. The general physical examination helps us to identify the severity of bleeding, subtle clues to the underlying cause for the bleeding, and the window for a few systemic diseases that are associated with GI bleeding. The examination should be rapid, focused, and specific to GI bleeding **(Table 5)**.

Systemic Examination (Gastrointestinal System)—Focused Abdominal Examination

Refer to Chapter 12 for detailed examination of gastrointestinal system.

Inspection:

- *Abdomen and perineum:* Look for dilated veins over the abdomen, spider angiomas, and umbilical protrusion (ascites). These are features of portal HTN as a cause. More common in upper GI compared to lower GI bleeding.

  Abdominal distension and visible peristalsis (may indicate obstruction or perforation). Organomegaly (focal distension—splenomegaly part of chronic liver disease and portal HTN, malignancies, etc.)
- *Surgical scars:* Postsurgical complications (recent or old) (suggests possible adhesions or postsurgical)

Ancillary:

- *Oral/nasogastric (NG) bleeding or coffee ground aspirate (indicate bleeding is not very recent):* Confirms UGIB. Helps to quantify and monitor the progress.
- *Stool manager:* Blood noticed—quantity, fresh/old.

Palpation:

- Tenderness (focal or generalized) with or without peritonitis features
- Gross organomegaly (rare), splenomegaly/malignancy (commonly gastric) uncommon to have growth, usually ulcerative.

TABLE 5: Summary of general physical examination of patients with gastrointestinal tract bleeding.

S. No.	*Domain*	*Eliciting of signs/symptoms*	*Comments*
1.	Head, face, and neck	• Confused state (when in shock/encephalopathy-hepatic) • Periorbital edema—liver failure • Eye—pale, icterus (severity of bleeding, liver pathology, DIC) • Fundus-uveitis/scleritis-IBD; CMV retinitis in disseminated infections, choroid tubercles: These are less common cause of upper GI bleeding • Mouth: – Bad breath – Bleeding gums, (coagulopathy: Favor DIC or liver failure—CLD) – Oral ulcers, thrush (possible gastric ulcers esophageal candidiasis) – Mucus pigmentation (Peutz-Jeghers syndrome, juvenile polyposis syndrome) • Cervical or supraclavicular lymphadenopathy—TB, GI malignancy	This gives a quick clue for the presence of possible systemic illness, disease severity
2.	Extremities	• Upper limb – Flapping termer • Lower limb—pedal edema (liver failure) – Pyoderma gangrenosum is associated with IBD, ulcerative colitis seen in some GI malignancies, IBD – Erythema nodosum—a tender red nodule is commonly seen in IBD • Fingers and palms – Clubbing-CLD—hepatic encephalopathy – Tripe palms (acanthosis palmaris) involve thickened, wrinkled skin mesh-like pattern with broadened ridges and deep groove on the palms and fingers	Quick examination of extremities helps to diagnose certain diseases that are associated with GI bleeding
3.	Trunk, back and perineum	• Rash–petechiae, ecchymosis, rash on trunk—herpes (simplex, zoster) • Genital ulcers—systemic diseases, infection • Spider naevi, gynecomastia—CLD • Telangiectasias • Blood in diaper • Scratch marks—itching	The perineal and back examination is often missed but important in patients with GI bleeding. Malena, active bleeding can be identified by examining the diaper
4.	Bedspace and documents	• Vasopressors, pantoprazole infusion, terlipressin, infusion • Blood sugars—hypo/hyperglycemias • NG drainage—Blood n bag (fresh vs altered blood), presence of bile rules out upper GI bleeding. Feculent material—possible intestinal obstruction: GI malignancy	A careful bedspace examination is helpful in getting the trends

This examination helps us to identify the severity of bleeding, subtle clues to the underlying cause for the bleeding and the window for few systemic diseases that are associate with GI bleeding.
(GI: gastrointestinal bleeding; CLD: chronic liver disease; IBD: inflammatory bowel disease; DIC: disseminated intravascular coagulation; CMV: cytomegalovirus)

- *POCUS and liver sonography:* Ultrasonography is an extension of examination—portal vein diameter, Doppler, organ size, and ascites are commonly noted. See the following text for details.

Percussion/Auscultation

- Confirmation of ascites through fluid shift remains the only specific feature to evaluate whilst other findings may not be relevant for the critically ill. For most other findings, ultrasound is easy and specific.
- Bruit—hardly done in ICU to assess for collaterals (portal HTN)

Per rectal examination (using surgical expertise if deemed necessary)

- Internal or external hemorrhoids
- Ulcers
- Quantity and quality of blood in the rectum (melena vs. fresh blood)/palpable masses
  - Melena (dark, tarry stools typically indicative of upper GI)
  - Fresh blood or maroon stools typically indicative of lower GI
  - Palpable mass—malignancy

- *Proctoscopy:* Proctoscopy where possible with surgical help (assessing for inflammatory or malignant causes).

Unlike clinical assessments which were based on classical teaching, most ICU admissions have already been seen, assessed and/or investigated by other clinicians. The newer ICU assessments not only involve clinical examination but also use significant adjuvants such as POCUS assessment and/or point-of-care testing (POCT).

Point-of-care ultrasound

Use of bedside ultrasound to assist with clinical assessment as noted earlier for shock and reassessment postresuscitation. It could also be used to find features that may aid in the diagnosis of chronic pathologies (liver cirrhosis).

Point-of-care testing

- ABG (for acidosis, lactate, blood urea, and hemoglobin)
- *Point-of-care full blood count:* Hemoglobin and platelet monitoring
- Point-of-care international normalized ratio (INR)/activated clotting time (ACT) to evaluate coagulopathy
- C reactive protein/high sensitivity-C reactive protein.

CLINICAL SCORES AS ADJUNCTS

Upper Gastrointestinal Bleeding

Before considering or admitting one to the ICU, they are usually assessed in an outpatient setting, an inpatient setting, a postendoscopy procedure, or in an ER. This could be subjective but due to the availability of multiple scoring systems, this can be made more objective. These scoring systems where clinical variables and/or investigations and /or endoscopy findings are used to risk stratify one for intervention, mortality, ICU admissions, rebleeding risk, or likely hospital stay, etc. Despite the availability of multiple scores, the accuracy is poor without the inclusion of the endoscopic features apart from many lacking external validation (including resource-limited settings) making their utility limited. But their use predominantly where clinical variables and POCT results are included might improve the triage technique. Rockall score, Glasgow–Blatchford Score (GBS), AIMS65, and the T-scores use only clinical or clinical and POCT results to risk stratify patients. The current broad recommendation to undertake an endoscopy with or without colonoscopy, within 24 hours of admission to the ICU of an UGIB patient, suggests the failure of scoring systems to appropriately triage the procedure requirement to date.

Coming to admission or evaluation of patients who already underwent endoscopy, compared to the Rockall score, the Forrest classification, which is entirely based on endoscopy findings, is likely to accurately risk stratify patients who may need ICU monitoring with respect to the risk of rebleeding or death.

Unlike their extensive use in pre-ICU settings, implications for decision-making, once a patient is admitted to the ICU, is limited apart from their rough estimate of mortality risk but not improving the available scoring systems such as sequential organ failure assessment (SOFA) or acute physiology, age, and chronic health evaluation (APACHE).

Lower Gastrointestinal Bleeding

Use of score to risk stratify LGIB for adverse outcomes: ABC score, NOBLADS, Sengupta score, BLEED, Brimingham score, severe acute LGIB (SALGIB) score, and HAKA score. Strate score which uses clinical information is more likely to inform about the need for intervention for LGIB.

SUMMARY

- In majority of the GI bleeding cases, it is the combined effect of the underlying illness and the blood loss that will make a patient to be triaged for and admitted to the ICU.
- Structured approach is more likely to focus efforts on appropriate causes and in turn investigation.
- Causes that require ICU admission are different from causes that are seen in less acutely-ill patient population.
- Focused clinical examination for upper and lower GI bleeding is varied and will enhance general examination of the critically ill.
- Shock assessment and resuscitation should concurrently be undertaken whilst the evaluation for the source is underway.

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Chapter

22

How will I Examine a Patient with Acute Gastrointestinal Tract Dysfunction/Acute Abdomen?

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ABSTRACT

Acute gastrointestinal dysfunction is often a problem faced in intensive care unit (ICU) patients. Abdominal pain or acute abdomen could be the initial primary presentation of a critical illness or could develop as a consequence of an already existing critical illness. The latter is usually a consequence of multiorgan dysfunction syndrome. In ICU patients, classical signs are often masked and make the diagnosis difficult. Identifying the cause through systematic clinical examination combined with patient history, laboratory tests, and imaging studies aids in early detection and helps in appropriate management. Point-of-care ultrasound at the bedside has further hastened the time to diagnosis. In this chapter, we learn about a focused approach to the clinical examination of the acute abdomen through a case scenario.

INTRODUCTION

Acute gastrointestinal dysfunction is common in intensive care unit (ICU) patients. Many have pain in abdomen, vomiting, distension, and altered bowel movements. The examination of the gastrointestinal system in ICU patients is often done for pain in the abdomen. Abdominal pain or acute abdomen could be the initial primary presentation of a critical illness or could develop as a consequence of an already existing critical illness. The latter is usually seen as part of multiorgan dysfunction syndrome. General or specific abdominal signs mostly help in the evaluation of the cause of acute abdominal dysfunction. In ICU patients, these classical signs are often masked and make the diagnosis difficult. Identifying the cause through systematic clinical examination combined with patient history, laboratory tests, and imaging studies aids in appropriate management. In this chapter, we learn about a focused approach to the clinical examination of the acute abdomen.

CASE SCENARIO

A 64-year-old gentleman presented to the emergency department with abdominal pain, nausea, and breathlessness. He is a known case of chronic obstructive pulmonary disease (COPD) with history of (H/o) of chronic smoking. Physical examination revealed abdominal distension with periumbilical guarding. He also has skin mottling over his legs. His oxygen saturation (SpO_2) was >90% with oxygen supplementation. He is confused but hemodynamically stable. Arterial blood gas (ABG) showed elevated lactates. Computed tomography (CT) scan of the abdomen with angiogram revealed a superior mesenteric artery thrombus with significant thrombosis of the aorta extending down to the iliac and femoral vessels. After initial resuscitation, he was initiated on systemic anticoagulation. Peritoneal signs were absent, and embolectomy was performed by the vascular surgeons. He was transferred to ICU for further management.

Abdominal pain can be benign needing only symptomatic treatment or may be an indicator of a life-threatening situation requiring surgical intervention. When it is severe, a rapid and focused evaluation of the cause helps in the quick identification of the problem.

Differential Diagnosis

Pain is an important symptom that may lead us to the clues for diagnosis. The type of pain, duration, exact location, time of onset, and severity should be evaluated. Often acute abdomen presents with diffuse abdominal pain and tenderness. In elderly patients or patients with multiorgan dysfunction, these features are very subtle and

TABLE 1: Differential diagnosis of pain abdomen based on the location.

Location of pain	***Differential diagnosis***
Right upper quadrant	• *Pulmonary:* Pneumonia and pulmonary embolism • *Biliary:* Cholecystitis, cholelithiasis, and cholangitis • *Colonic:* Colitis and diverticulitis • *Hepatic:* Mass, abscess, and hepatitis • *Renal:* Pyelonephritis and renal stone
Epigastric	• *Gastric:* Esophagitis, gastritis, and peptic ulcer • *Biliary:* Cholecystitis, cholelithiasis, and cholangitis • *Pancreatic:* Mass and pancreatitis • *Cardiac:* Myocardial infarction and pericarditis • *Vascular:* Mesenteric ischemia and aortic dissection
Left upper quadrant	• *Cardiac:* Angina, myocardial infarction, and pericarditis • *Gastric:* Esophagitis, gastritis, and peptic ulcer • *Pancreatic:* Mass and pancreatitis • *Renal:* Nephrolithiasis and pyelonephritis • *Vascular:* Aortic dissection and mesenteric ischemia
Right lower quadrant	• *Renal:* Renal stone and pyelonephritis • *Colonic:* Appendicitis, colitis, diverticulitis, IBD, and IBS • *Gynecologic:* Ectopic pregnancy, fibroids, ovarian mass, torsion, and PID
Left lower quadrant	• *Gynecologic:* Ectopic pregnancy, fibroids, ovarian mass, torsion, and PID • *Colonic:* Colitis, diverticulitis, IBD, and IBS • *Renal:* Nephrolithiasis and pyelonephritis
Periumbilical	• *Colonic:* Early appendicitis • *Gastric:* Esophagitis, gastritis, peptic ulcer, and small bowel mass or obstruction • *Vascular:* Aortic dissection and mesenteric ischemia
Suprapubic	• *Colonic:* Appendicitis, colitis, diverticulitis, IBD, and IBS • *Gynecologic:* Ectopic pregnancy, fibroids, ovarian mass, torsion, and PID • *Renal:* Cystitis, nephrolithiasis, and pyelonephritis
Abdominal wall	Herpes zoster, muscle strain, and hernia
Extra-abdominal causes	DKA, pneumonia, pulmonary embolism, and herpes zoster
Other conditions (not well localized)	Drugs/toxin, narcotic withdrawal, sickle cell crisis, porphyria, IBD, heavy metal poisoning, malignancy, toxic megacolon, mesenteric lymphadenitis, SLE, rectus sheath hematoma, hypercalcemia, and insect bites

(DKA: diabetic ketoacidosis; IBD: inflammatory bowel disease; IBS: irritable bowel syndrome; PID: pelvic inflammatory disease; SLE: systemic lupus erythematosus)

may be missed. The clinical abdominal examination might be highly deceptive in patients with acute abdomen who are already having cirrhosis with ascites. The location of pain should guide further evaluation **(Table 1)**.

Life-threatening conditions that need immediate diagnosis and management are:

- Myocardial infarction
- Perforation of gastrointestinal tract
- Abdominal aortic aneurysm (AAA)
- Acute intestinal obstruction
- Mesenteric ischemia
- Splenic rupture
- Volvulus
- *Obstetric causes:* Ectopic pregnancy and placental abruption.

History Taking

History eliciting combined with physical examination is crucial before analgesia and/or sedation. This should not stop from treating the pain. Once the location of the pain is known, further detailed history about the onset, duration, severity of pain, and factors increasing or decreasing should be asked. Other associated symptoms should be considered focusing on differential diagnosis.

While few symptoms carry the highest positive predictive value to rule in few conditions, few symptoms considered important in the diagnosis may carry very low positive predictive value.

We should consider the patient's age, sex, past medical and surgical history, and medications to characterize the pain as precisely as possible.

- *Age:*
    - Elderly groups are more:
        - Likely to have more severe manifestation
        - Atypical symptoms are more
        - Present with different symptoms for the same problem
        - Previous medications like steroids may mask the symptoms
        - Likely to present with hypothermia than the younger population.
- *Sex:*
    - Females:
        - Genitourinary causes should be considered.
        - Pregnancy: Consider both pregnancy related and nonpregnancy related.
        - Pregnant patients may not show classical peritoneal signs as the stretching peritoneal cavity is more desensitized. This makes physical examination and interpretation of findings difficult.
        - Childbearing age: Pregnancy status should be checked.
        - Reproductive age group: Unsafe abortions and urinary tract infections are often missed.
    - Males:
        - Excess workout or newly started exercise may present as abdominal pain, whereas the actual problem may be in abdominal wall muscles.
        - Genitourinary causes should be evaluated.
        - Perform testicular examinations in men with pain in the lower half of the abdomen.

Preexisting medical and surgical conditions and medications:

- Ischemic heart disease with atrial fibrillation: Increase risk of gut ischemia and stroke
- Previous abdominal surgeries: Intestinal obstruction
- Human immunodeficiency virus (HIV): Risk of opportunistic infections
- Nonsteroidal anti-inflammatory drugs (NSAIDs): Risk of acute kidney injury (AKI)
- Chronic liver disease (CLD): Spontaneous bacterial peritonitis (SBP).

Social History

- *Smokers:* Risk of bladder cancer
- *Alcohol:* CLD and pancreatitis
- *Drug abuse:* Nonspecific acute abdominal pain

Occupational and Travel History

- *Occupational exposures to toxins or chemicals:* Nonabdominal causes
- *Recent travel:* Infectious causes

Examination

General Examination

Focused examination:

- In the patient discussed earlier, we should start with ABCDE approach.
    - A—airway: He is hypoxic requiring supplemental oxygen with patent airway.
    - B—breathing: He is tachypneic with no significant distress.
    - C—circulation: Stable blood pressure with tachycardia.
    - D—disability: He is confused but moving all limbs and obeying simple commands.
    - E—exposure: He has distended abdomen with skin mottling changes.

    If airway and hemodynamic parameters are unstable then the focus lies on the stabilization of the patient.

    He has H/o nausea and vomiting, vague abdominal pain, and distention. Elderly patients with vague abdominal pain with elevated lactates should always give raise to suspicion of gut ischemia.
- In any such similar scenario, we get information from focused general examination.

Vitals signs:

- *Tachycardia:*
    - It indicates fever or sympathetic activity due to pain.
    - Fever indicates infection. Absence of fever does not rule out any of these things. Especially elderly or immunocompromised may not mount fever response.
    - Tachycardia with orthostatic hypotension: Hypovolemia
- Hypotension indicates shock of any origin like sepsis, hypovolemic, obstructive, and distributive.

- *General physical examination:* Apart from routine examination, focused gastrointestinal (GI) system examination helps us to narrow down the diagnosis.
    - Routine examination:
        - Pallor, icterus, cyanosis, clubbing, koilonychia, lymphadenopathy, and edema
        - Oral cavity: Ulcers
    - Focused GI examination: Patients with acute abdomen may present with GI bleed, obstruction, perforation, intra-abdominal sepsis, and raised intra-abdominal pressure. Certain problems may present with typical presentations and straight forward while few may not.
        - Renal colic patients will have severe pain and agitated.
        - Peritonitis patients may lie still.
        - Pancreatitis patients—distended abdomen with respiratory distress with signs of SIRS
    - We also need to look at:
        - Look at nasogastric (NG) tube, indication, NG tube output, stool frequency, other drains, and tubes placed
        - Intra-abdominal pressure monitoring

Systemic examination:

- *Inspection:* Adequately expose the patient after informed consent **(Fig. 1)**. Have a female nurse while examining a female patient.
    - Shape: Flaccid, distended
    - Umbilicus shape and position: Umbilicus shape and position may give clues about lower versus upper abdominal pathology, hernias, obesity-related issues, and massive ascites.
    - Skin color: Discoloration of the left flank is associated with acute hemorrhagic pancreatitis (Grey Turner sign)
    - Abdominal distention with spider angiomata: Cirrhosis
    - Signs of previous surgeries: These signs may suggest the possibility of adhesion-related issue.
- *Auscultation:*
    - Bowel sounds: Normally heard as 2–12 medium-pitched gurgles per minute.
    - Peritonitis: Absent bowel sounds over 2 minutes
    - Inflammation of GI tract: Hyperactive medium-pitched bowel sounds
    - Bowel obstruction: Periodic rushes of high-pitched "tinkling" bowel sounds or the complete absence of bowel sounds

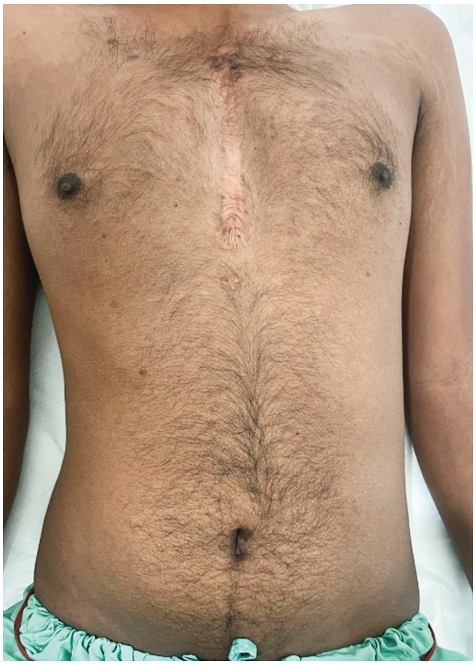

Fig. 1: Expose the patient from nipple level to pelvic area for inspection.

    - A bruit—AAA
    - Critically ill patients: Often the bowel sounds are sluggish.
- *Palpation* ***(Fig. 2)****:*
    - Start from the area away from pain.
    - Check clockwise or anticlockwise to reach the maximal area of pain.
    - Deeper palpation may be required to identify other abnormalities, such as hepatomegaly, splenomegaly, aortic dilatation, or signs of a retrocecal appendix.
    - Findings like tenderness, rebound tenderness, and rigidity guide us.
    - More maneuvers to elicit somatic signs can be performed once the area of maximal tenderness is localized.
- *Ultrasonography (USG):* Adding bedside USG to the examination in the setting of critically ill patients like FAST (focused assessment with sonography for trauma) and POCUS (point-of-care ultrasound) helps to detect:
    - Ascites, renal, and gallbladder pathologies
    - Peristaltic movements
    - Can pick up even smaller amounts of ascites.
    - Can differentiate between air or ascites as the cause for abdominal distention.

- Can help assess the cause of shock.
- May guide us to consider further imaging like CT scan.
- It can even help us to tap fluid bedside and identify the type of fluid.

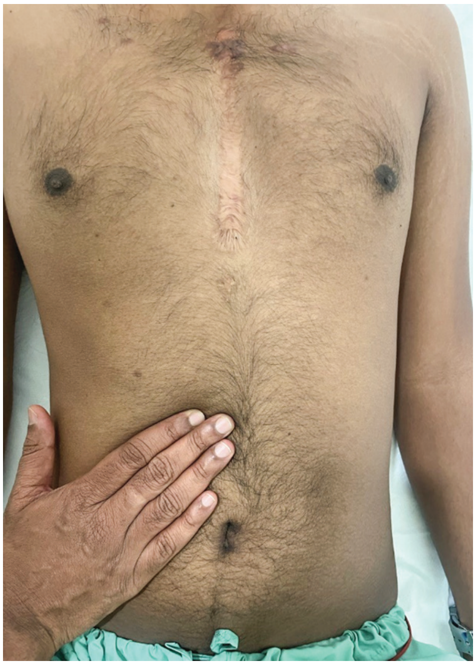

Fig. 2: Palpation of abdomen.

- Certain signs or maneuvers that can help in the diagnosis are mentioned in **Table 2**.
- *Percussion:*
    - Air versus fluid
    - Loss of liver dullness
    - Urinary bladder dullness
- Cardiovascular system and respiratory system examination should be performed without fail as upper abdominal pain could be due to:
    - Pneumonia or acute coronary syndrome (ACS)
    - Empyema.

Diagnostic Testing

Diagnostic testing should always be used as adjuncts to clinical examination.

Laboratory Tests

Laboratory tests should only be ordered to assess a patient with an acute abdomen of unclear etiology, and immunosuppressed or older adults. Otherwise, it may not be necessary in an otherwise healthy adult. However, if the patient is critically ill, basic organ function tests are always required to look for another organ dysfunction. Depending on the differential diagnosis, more specific tests can be performed.

- *Patient with epigastric pain:* Isolated elevation of lipase levels with normal amylase levels may not

TABLE 2: Signs or maneuvers that help in the diagnosis.

Murphy's sign	In patients with cholecystitis (present only in 65% of adults with cholecystitis and is particularly unreliable in older patients)
Carnett's sign	Increases pain when the supine patient tenses the abdomen by lifting the head and shoulder indicating pain from the abdominal wall
Psoas sign	In patients with appendicitis
The psoas, obturator, and Rovsing signs	These are less sensitive but have good specificity for acute appendicitis
Signs of rigidity and rebound tenderness	These have limited sensitivity and specificity, but a rigid abdomen is always a concern
Rectal and pelvic examinations	In patients with lower abdominal and pelvic pain. It may reveal fecal impaction, a palpable mass, or occult blood in the stool
Courvoisier's sign	Obstructive jaundice: If the gallbladder is palpable, it is not due to gallstones (Courvoisier's law)
Kehr sign	Splenic rupture: Pain elicited in left shoulder. It is a classic example of referred pain due to irritation of the diaphragm
Tenderness and fullness on the right side of the rectum	Suggest a retrocecal appendix
A pelvic examination	Presence of cervical motion tenderness and peritoneal signs suggests high likelihood of ectopic pregnancy or other gynecologic complications like salpingitis or a tubo-ovarian abscess

indicate pancreatitis and may suggest need for more investigations.

- Check urine pregnancy test in women of childbearing age with abdominal pain
- Liver function test in CLD
- Urine analysis in flank pain.

The clinical evaluation could be extremely deceiving in cases of secondary peritonitis occurring in cirrhotic patients with ascites. Runyon criteria have been used to evaluate the ascitic fluid in such cases considering the protein, glucose, and lactate dehydrogenase (LDH) values. If two of the given three criteria are present (i.e., protein >1 g/dL, glucose <50 mg%, and LDH > upper limit of serum level), secondary peritonitis is very likely. Diagnosis of secondary peritonitis is further supported by the polymicrobial nature of the ascitic fluid. Carcinoembryonic antigen (>5 ng/mL) or alkaline phosphatase level (>240 units/L) in the ascitic fluid have been found to be more sensitive and specific markers for secondary peritonitis in cirrhotic as well as noncirrhotic ascites.

Imaging Studies

Decisions for imaging studies are generally based on the location of the abdominal pain. It also depends on the location of the patient, access, and stability of the patient for transfer. Bedside X-ray is nowadays easy but has a lot of limitations. Bedside ultrasound usage not only by radiologists but also by intensive care physicians had made the diagnosis quicker. Many times, patients may be extremely sick to shift for CT scans. Judicious use of imaging wherever appropriate helps in rapid diagnosis.

- *Plain radiograph of the abdomen:*
  - It is useful to detect bowel perforation, obstruction, and foreign bodies.
  - Only a small percentage of plain radiographs are abnormal and cannot exclude these disorders.
  - Bowel perforation: It shows air under the diaphragm. Free air is seen only in two-thirds of cases of gastroduodenal perforation and in one-third of cases with perforation of the distal small bowel or large bowel. In walled-off perforations, the sensitivity further reduces.
  - Bowel obstruction: Plain X-rays show a curvilinear array of small gas bubbles ("string of beads" sign), which is pathognomonic for small bowel obstruction. Small gas bubbles collected between the valvulae conniventes floating in a fluid-filled bowel give the above appearance. Small bowel <3 cm, large bowel <6 cm, and caecum <9 cm is normal. Plain radiographs are up to 69% sensitive and 57% specific to diagnose obstruction.
  - Mesenteric ischemia: Findings like ileus, "thumbprinting", and intramural air (pneumatosis intestinalis)
- *Ultrasound:*
  - Nowadays ultrasound is widely available in the ICUs and emergency room (ER). Easy to perform bedside by ER physicians, ICU physicians, or radiologists. As it can be rapidly performed, it is the initial choice of investigation in unstable patients. The radiologist performed abdominal examination will be more comprehensive, whereas the intensivist performed examination is more specific and focused.
  - USG can give quick information about hemodynamic status, gallbladder disease, pancreatitis, free fluid in the peritoneum, traumatic hemoperitoneum, ruptured ectopic pregnancy, and AAA leak or rupture, evaluating ovarian, uterine, and scrotal pathology, renal colic (hydronephrosis may be seen), venous thrombosis, and acute pulmonary embolism.
  - Nodular liver surface, round edge, and hypoechoic nodules in liver parenchyma which represent regenerative nodules of cirrhotic liver point toward liver cirrhosis. Detection of hypoechoic nodule >10 mm is important in the early diagnosis of hepatocellular carcinoma. USG of gallbladder with focus on main problems such as cholelithiasis, gallbladder wall thickening, and pericholecystic fluid helps in identification of source of sepsis like stones, sludge, thickening of the GB wall (>3 mm), pericholecystic fluid, or dilated common bile duct (>4 mm).
  - In case of AKI, bedside USG helps to assess empty bladder with appropriate Foleys location or distended bladder, suggesting catheter blockage prompts change of catheter. Postrenal obstruction leading to hydroureteronephrosis (HUN) needs to be addressed immediately. A normal kidney has a hyperechoic medulla and relatively hypoechoic cortex. With increasing degrees of obstruction, the collecting system will begin to dilate, extending up into the minor and major calyces, and eventually thinning the cortex of the kidney. This can be

graded from mild to severe. All these findings help in identifying the important causes of intra-abdominal pathology.

- *CT scan:*
  - CT is the choice of investigation in a patient with undiagnosed acute abdomen. If the patient is stable to undergo CT, it outperforms plain radiography or ultrasound in the information that can be provide.
  - Intravenous (IV) contrast: It facilitates the diagnosis of potential etiologies in patients presenting with nonlocalized abdominal pain. With the advent of very good quality images of CT scan routine use of IV contrast is now questionable. But IV contrast carries a significant role in certain conditions.
  - CT angiography: It helps in visualizing the mesenteric vasculature and findings consistent with bowel infarction. If bowel ischemia is not the cause of abdominal pain, CT angiography helps in finding out other pathologies. It is also useful to identify the source of gastrointestinal bleeding with the ability to detect bleeding rates of 0.3 mL/min.

SUMMARY

- Acute gastrointestinal tract dysfunction/acute abdomen can present as primary problem or may be a manifestation of multiorgan dysfunction in critically ill patients.
- Timely identification of the problem is important, especially in sick comatose patients.
- A sequential and systematic approach to examination combined with appropriate history taking along with other supports like laboratory evaluation, imaging, and bedside ultrasound helps critical care physician to identify the problem on an urgent basis.
- Examination in critically ill patients should emphasize a quick more focused approach than the stable ward patient.

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Chapter

23

How will I Examine a Patient with Acute Chest Pain in the ICU?

Sristi Patodia, Raymond Dominic Savio

ABSTRACT

This chapter focuses on the comprehensive assessment of patients presenting with acute chest pain in the intensive care unit (ICU). Acute chest pain is a critical symptom that necessitates prompt and precise evaluation to determine its etiology, which can range from benign to life-threatening conditions. The chapter outlines a systematic approach to patient evaluation, integrating clinical history, physical examination, and diagnostic testing. Key areas covered include the differentiation of cardiac and non-cardiac causes of chest pain, the utilization of advanced imaging modalities, and the interpretation of laboratory tests such as cardiac biomarkers. The chapter also emphasizes the importance of a multidisciplinary approach, involving collaboration with cardiologists, pulmonologists, and other specialists, to ensure accurate diagnosis and optimal patient management. By providing a detailed protocol for the assessment of acute chest pain in the ICU, this chapter aims to enhance clinical decision-making and improve patient outcomes.

INTRODUCTION

William James Mayo once said, “Sometimes I wonder whether today we take sufficient care to make a thorough physical examination before our patient starts off on the round of the laboratories, which have become so necessary that oftentimes we do not fully appreciate the value of our five senses in estimating the condition of the patient”.

CASE SCENARIO

A 54-year-old male, who is diabetic, hypertensive, and a smoker, had a slip and fall and sustained a fracture to the right tibia for which he had been confined to bed for a week. He has now undergone an open reduction and internal fixation for the same and has been received in the ICU for postoperative monitoring. 1 hour after arrival, the patient complained of chest pain. Further history suggests that the chest pain was sudden in onset and is around the center of his chest. The patient appears tachypneic and his oxygen saturation (SpO_2*) has dropped from 98% in room air to requiring 10 L of supplemental oxygen. His heart rate (HR) has also increased to 124 beats/min from 101 beats/min (regular) and his blood pressure (BP) is now 90/50 mm Hg over the previous value of 120/70 mm Hg.*

It is very imperative at this stage to commit to a working diagnosis in order to proceed further with a successful strategy. A blind approach will only further complicate the course of illness and add to treatment delay and cost of care. It would be an interesting mind game to set out a list of differential diagnosis and think of factors favoring or contradicting them while at the same time performing the initial stabilization. It needs to be emphasized that while systematic examination is prudent, it has to happen simultaneously with initial resuscitation and stabilization.

Given the acute onset of chest pain with respiratory and circulatory failure one should be quickly prompted to think of the following life-threatening possibilities.

- *Acute pulmonary embolism (PE):*
  - Background history of immobilization
  - Fracture surgery
  - Respiratory and circulatory failure
- *Acute coronary syndrome (ACS):*
  - Age and risk factors
  - Perioperative risk and stress
  - Cardiorespiratory failure could imply a significant left ventricular (LV) dysfunction.
- *Pneumothorax:*
  - Unlikely from initial trauma because of the time delay

- Breathlessness would dominate over chest pain.
- To consider iatrogenic causes, e.g., central line insertion/barotrauma

History and Symptomatology

To elicit a proper history and do a focused examination, one must know what to look for. After all what the mind does not know, the eyes cannot see.

Chest pain is a common presenting complaint in the emergency department and intensive care unit. However, the causes of chest pain are many and can be a manifestation of multiple organ systems. Some of these causes could be acutely life-threatening **(Box 1)**. There could be a myriad of other causes for not-so-acute chest pain **(Table 1)**. While these may not fit our current patient scenario, it is worthwhile subconsciously ruling them out.

History

A good clinical examination begins with a great history. "Medicine is a science of uncertainty and an art of probability", said William Osler. A good history is invaluable in increasing the probability of a correct diagnosis. The patients' history will often help to narrow down to the most likely organ system involved, thus allowing for a focused system examination.

When eliciting the history of any pain, the "must ask" questions should seek for details regarding *S*ite, *O*nset, *C*haracter, *R*adiation, *A*ggravating factors, *T*ime course, *E*xacerbating and relieving factors and *S*everity. This is often covered by the mnemonic SOCRATES. Once we have the overall description of the pain, it becomes easy to narrow down toward certain etiologies based on specific characteristics as shown in **Table 2**.

BOX 1: Life-threatening causes of acute chest pain (The Big Six).

1. Acute coronary syndrome
2. Pulmonary embolism
3. Tension pneumothorax
4. Acute aortic dissection
5. Pericardial tamponade
6. Mediastinitis (commonly esophageal rupture)

TABLE 1: Other causes of chest pain.

Thoracic causes	***Abdominal causes***	***Miscellaneous causes***
Chest contusion	Peptic ulcer disease	Somatization and panic
Tietze syndrome	Cholecystitis	Bornholm syndrome
Pneumonia	Cholangitis	Herpes zoster
Pleurisy	Biliary colic	Bone metastasis
Esophagitis/GERD	Hepatitis	Referred pain
Pneumomediastinum	Pancreatitis	Da Costa syndrome
Valvular heart disease	Aortic aneurysm	Migraine

(GERD: gastroesophageal reflux disease)

Examination

The clinical examination of a patient should be systematic from head to toe and with attention to teasing out the primary diagnosis from the list of differentials arrived at from history. This includes a general and a systemic examination.

General Physical Examination

A thorough general physical examination begins with the assessment of patient's level of consciousness [Glasgow Coma Scale (GCS) and orientation], examination for common pointers to specific disease process, and measurement of vital signs such as temperature, pulse rate, BP, SpO_2, and respiratory rate (RR).

The assessment for GCS is crucial to any disease entity and more so in a setting of chest pain wherein a drop in GCS could imply a significant reduction in global brain perfusion, an ischemic state from cardioembolism, a cardiac arrest, metabolic encephalopathy, etc.

This is followed by a careful head-to-toe examination for any clue/pointers to specific disease entities. The never-to-miss findings are suggested by the mnemonic PICCLE, which is expanded as *P*allor, *I*cterus, *C*yanosis, *C*lubbing, *L*ymphadenopathy, and *P*edal *E*dema. While there could be several system-specific reasons for the above findings, in a setting of chest pain, the following are to be borne in mind although possibilities are abound.

- *Pallor:* This could be from anemia which can precipitate a myocardial infarction (MI) due to supply–demand mismatch or from significant vasoconstriction as happens during a cardiogenic shock. Alternately, anemia and therefore pallor can also result from blood loss, e.g., ruptured LV/aneurysm.
- *Icterus:* This is a sign of metabolic failure of liver possibly arising from a congestive hepatopathy as a consequence of right heart failure, constrictive

TABLE 2: Differential diagnosis based on history/presenting features.

Disease	***Symptoms onset***	***Character***	***Radiation***	***Severity***	***Associated factors***
Acute coronary syndrome	Acute/hyperacute	Crushing/feeling of elephant over the chest	To ipsilateral arm, jaw, or back	Very severe	Aggravates on exertion, relieved on rest
Aortic dissection	Acute	Central chest pain—tearing feeling	To back	Severe	Presence of end-organ ischemia such as a loss of consciousness, intestinal ischemia
Pulmonary embolism	Acute/subacute				Breathlessness often predominates
Pericardial tamponade	Acute/subacute	Central chest pain		Severe	Relieved on leaning forward, rapid progression to shock
SVC syndrome	Subacute	Feeling of tightness over chest and face with facial swelling and breathlessness		Moderate to severe	Characteristic facial swelling, engorged neck veins
Mediastinitis	Acute/subacute	Retrosternal chest pain		Moderate	Associated with fever/cough or recent upper GI surgery
Tension pneumothorax		Breathlessness predominates			Similar to tamponade, rapid progression to shock
Boerhaave's syndrome	Acute	Retrosternal pain/burning		Severe	Classical history of retching/vomiting

(GI: gastrointestinal; SVC: superior vena cava)

pericarditis, cardiac cirrhosis, etc. A shock liver from impaired cardiac output can also be one of the reasons for liver dysfunction.

- *Cyanosis:* Extreme hypoxia in the setting of cardiogenic pulmonary edema, PE, or congenital heart diseases could amount to cyanosis when the level of deoxygenated hemoglobin exceeds 5 g/dL. Peripheral causes include vasoconstriction from cardiac shock or use of vasopressors, venous stasis, etc. While hemoglobinopathies are additional causes of cyanosis, these are not within the perspective of chest pain.
- *Clubbing:* Clubbing in a person presenting with chest pain should prompt a search for causes such as chronic obstructive pulmonary disease (COPD) (emphysema) presenting with a spontaneous pneumothorax, lung malignancy with pleural involvement, rib metastasis, lung abscess, etc.
- *Lymphadenopathy:* In a setting of chest pain, significant cervical lymphadenopathy could be a pointer for an infective focus such as a retropharyngeal abscess with or without mediastinitis.
- *Pedal edema:* The cause for this could be attributed to many organ system dysfunctions; however, in our scenario, this should raise the possibility of congestive heart failure.

It should be remembered that the above discussion is not exhaustive and just an example for focused interpretation of a general finding. Additional assessment should include facial features such as flushing, diaphoresis, flaring of alae nasi, and pursed lip breathing.

Vital signs: Overall, these signs hold great significance in gauging the urgency, especially in patients complaining of chest pain. Derangements in any of these five vital signs will suggest a gamut of possibilities, and it is very essential to conceive the underlying pathophysiological process in mind rather than merely make a document of numbers. Other important features such as anxiety, distress, diaphoresis, and blushing associated with pain or sympathetic stimulation are also to be actively looked for and correlated to the underlying disease state.

Pulse: Examination of pulse is incomplete unless one carefully assesses the rate, rhythm character, and symmetry in all four limbs. It will be preferable in a nonemergency setting to additionally assess for nature of vessel wall, presence of bruits, etc. There could be a wealth of information gathered from a careful assessment of pulse with pointers toward cause and consequence of chest pain. While a tachycardia may be physiological arising from pain, anxiety, or apprehension, it could also be from a significant supraventricular tachyarrhythmia (SVT) or a life-threatening ventricular tachycardia (VT). Atrial fibrillation (AF) can be diagnosed by identifying a discrepancy between HR and an irregular pulse rate. Similarly, bradycardia could be the result of atrioventricular blocks that are frequently seen with inferior wall MI or from drug therapy. Assessment of pulse volume could give us an idea about a prevailing low or high cardiac output state. Pulsus paradoxus, which refers to an exaggerated inspiratory weakening of pulse, could be a very important finding in a setting of chest pain and can be seen in several conditions such as severe asthma, cardiac tamponade, and PE. Several other characteristics of a pulse are traditionally described but often overlooked in an emergency setting.

Blood pressure: This is a very significant vital sign in a patient with chest pain and should be checked in both arms. We cannot stress enough the importance of appropriate size and positioning of the cuff. Hypertension could be physiological, de novo from an acute anterior wall MI, accelerated hypertension, etc. Likewise, hypotension could be the result of a cardiogenic or obstructive shock in a setting of chest pain. Both these have to be managed with urgency to prevent additional complications.

Always check for all peripheral pulses and BP in all four limbs as unequal BP or a pulse deficit could be pointers toward acute aortic dissection.

Respiratory rate: Changes in RR, akin to HR, could be from physiological or pathological reasons and could be a manifestation of both cardiac and respiratory events.

Temperature: Fever may raise the suspicion of an ongoing infection precipitating the chest pain such as infective endocarditis, empyema, and synpneumonic effusion. Hypothermia can also be a sign of ongoing sepsis or cardiogenic shock.

Oxygen saturation: Finger pulse oximetry has become a standard and most crucial part of any cardiorespiratory evaluation as it helps identify severity and therefore urgency.

System Examination

Inspection: The inspection of the chest always begins with adequate exposure, visualization of the neck, thorax, and concludes with a quick look over the abdomen and the back. While acute, life-threatening emergencies in the ICU seldom give us the time for a methodical examination, it cannot be stressed enough that these steps should run in a quick succession along with initial stabilization. Such a synchronous performance comes only with repeated bedside application and experience. A simplified but not exhaustive checklist for respiratory and cardiac system examination in a patient with chest pain is provided in **Tables 3 and 4**.

Neck: Conspicuous findings in acute situations may include a tracheal deviation to the opposite side as occurs in tension pneumothorax or to the same side as seen in

TABLE 3: Minimalistic summary of respiratory system examination in a patient with chest pain.

Inspection	General appearance, posture: • Neck—tracheal position, dilated veins and pulsations, scars • Chest—shape, respiratory rate, rhythm and pattern, bilateral symmetry, signs of increased work of breathing (WOB), scars, rash • Upper abdomen and back—scars, rash, and visible pulsations
Palpation	• Neck—to confirm inspection findings, check for significant lymph node enlargement • Chest—tenderness, symmetry of chest expansion
Percussion	To identify normal resonance in all areas versus dullness or hyperresonance
Auscultation	To identify normal (vesicular) versus abnormal (wheeze, crackles) breath sounds in all areas

TABLE 4: Minimalistic summary of cardiac system examination in a patient with chest pain.

Inspection	General appearance and features of specific phenotypes: • Neck—jugular venous pulsations • Chest—visible pulsations, precordium, and apex
Palpation	• Detailed assessment of pulse, blood pressure, and jugular venous pressure (JVP) • Apical pulse, palpation for precordial heave and thrills
Percussion	Not recommended in critical care examination
Auscultation	• Quality of S1 and S2, additional heart sounds (S3, S4, murmurs, opening snap, and pericardial rub) • Carotid, abdominal bruits

TABLE 5: Key examination findings in common cardiorespiratory emergencies.

	Inspection	*Palpation*	*Percussion*	*Auscultation*
Respiratory system				
Pneumothorax	Unilateral fullness, trachea pushed to the opposite side, decreased chest rise on the affected side	Confirm inspection findings	Hyperresonant note over affected area	Absent breath sounds
Pneumonia	No significant finding	May have tenderness over the involved lobe	Dull	Crepitations
Pleural effusion	Trachea pushed to the opposite side	Tenderness over the affected area	Dull	Absent breath sounds
Cardiovascular system				
Congestive cardiac failure	Apical impulse pushed down and out	Confirm inspection finding. The thrill may be present over the apical impulse		S4 may be heard
Cardiac tamponade	Engorged blood vessels, distended neck veins	Same as in the inspection	Dull note over the heart borders	Muffled heart sounds

atelectasis. The neck veins may be engorged in pericardial effusion with impending tamponade or PE.

Chest: A quick look over the chest wall should inspect for any visible scars, marks, pigmentation, dilated veins, and visible pulsations. More important would be the inspection of adequacy and equality of chest movements with respiration. This is paramount in our given scenario where there could be limited chest expansion either from pneumothorax or splinting of the chest because of pain. Try to visually locate the point of maximal impulse, known as the apex, which is typically located on the left fourth to fifth intercostal space.

The presence of surgical scars such as from a pneumonectomy, an old intercostal drain or a sternotomy can give valuable insight into the underlying disease process. Dilated veins, known as spider naevi, are often suggestive of chronic liver disease, although they can occur in any hyperestrogenic state. Visible pulsations, especially a precordial heave, are suggestive of right ventricular hypertrophy and dilatation.

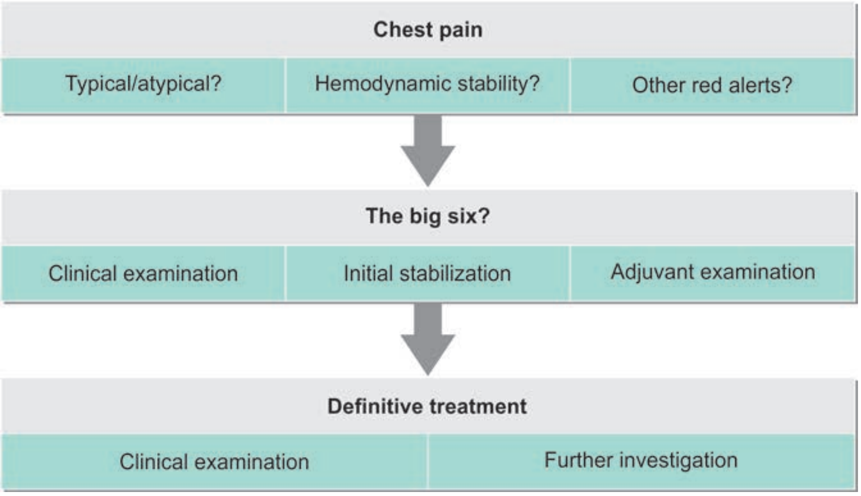
The location of the apex can be pushed down and out in LV hypertrophy. In diseases of the right ventricle, where there may be a large right ventricular hypertrophy, the left ventricle may be pushed posteriorly and the apex may be seen in the left parasternal border (most likely the right ventricular apex).

Palpation: Palpation is done to confirm all findings noted during inspection. In our scenario, this would include a careful confirmation of tracheal position and chest movements with respiration. The presence of any tender points is to be assessed carefully as this could point away from typical angina and possibly suggest a musculoskeletal etiology such as costochondritis and rib fracture. Additional findings to be looked for during palpation are as follows **(Table 5)**:

- *Crepitations:* These are suggestive of subcutaneous emphysema
- *Apex:* Location should be confirmed and then the apex should be palpated to note its character. A patient with a high cardiac output state (hyperdynamic circulation) will often have an apex that may occupy more than one intercostal space and may feel as a strong impulse lifting the examiner's finger with each beat.
- *Thrill:* It is a continuous palpable vibration that accompanies murmurs, and it is often referred to as the palpable murmur. The presence of a thrill almost always confirms that a murmur is pathognomonic.
- Palpation for aortic pulsation (abdomen—aortic aneurysm)
- S3 gallop—could indicate heart failure. S4 is also called the atrial gallop and could suggest LV dysfunction.

Percussion: While there is no doubt that clinical medicine has come a long way from traditional methods to gadget-based, goal-directed assessment, we cannot belittle the importance of conventional bedside examination, especially given the possibility of coupling findings with bedside point-of-care laboratory or imaging guidance. The ease and speed of identifying a very resonant or dull note

Flowchart 1: Evaluation of chest pain.



on percussion of the chest and therefore the likely diagnosis can only be acknowledged by the passionate clinician! Always ensure that percussion assessment is done on as much area of the chest as possible without wasting too much time on finer details. The most significant signs of percussion that are relevant in an ICU would be the hyperresonant note of pneumothorax and a dull note with local tenderness is suggestive of empyema.

Percussion of the heart borders will help to identify any enlargement of the chambers of the heart, but this is seldom practiced.

Auscultation: Auscultation should begin with the respiratory system, wherein one must listen for vesicular breath sounds bilaterally, in all areas of the chest. While it is preferable to examine all areas of the chest systematically, it may often be required to perform a rapid and limited assessment in a life-threatening emergency.

The absence of breath sounds could suggest a pneumothorax or any fluid in the pleural space. Breath sounds may also be absent in the case of a complete collapse of a lung on the affected side. It is imperative to actively search for a wheeze and crackles in all areas of the chest. This will help point toward airway and parenchymal pathologies, respectively.

This is followed by auscultation for heart sounds. This is done in the traditional five points for cardiac auscultation, namely the mitral, tricuspid, aortic, pulmonic, and Erb's points. In order to know the timing of the cardiac cycle, the apex must be palpated and auscultation started from there as the S1 (first heart sound) is loudest at this position. S2 (second heart sound) is best assessed from the aortic area. The quality of S1 and S2 is to be noted. A loud or muffled pattern signifies disease states. The quality of S1 is variable in AF. A loud A2 or P2 could signify the presence of systemic or pulmonary hypertension, respectively. The presence of S3 and S4 similarly needs to be noted as these could point toward ventricular dysfunction.

There are many murmurs that can be picked up but those significant from a critical care perspective are as follows:

- Any new onset murmur which was not present earlier, such as a mid-diastolic murmur that is heard in mitral regurgitation (MR) or a holodiastolic murmur heard in ventricular septal defect (VSD) is suggestive of complications of acute MI.
- Presence of changing murmurs, especially in a patient with a history of fever will be suggestive of infective endocarditis.
- The holosystolic murmur of acute tricuspid regurgitation is heard best at the Erb's point (left side of the sternum between third and fourth ribs).

One must also auscultate for pleural/pericardial rubs suggestive of fluid in the respective spaces. A schematic representation of the above description is depicted in **Flowchart 1**.

Adjuvant Examination

Clinical examination will seem quite incomplete without complimenting them with the many point-of-care bedside tests that are currently available **(Fig. 1)**, which increase

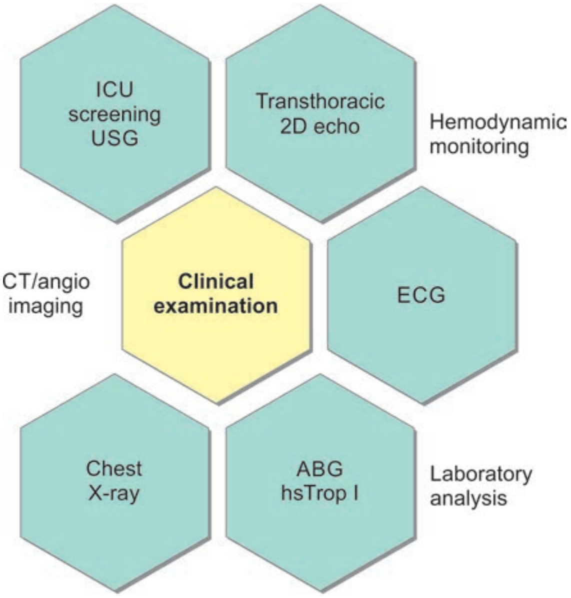


Fig. 1: Essential adjuvants to clinical examination. (ABG: arterial blood gas; CT: computed tomography; ECG: electrocardiography; ICU: intensive care unit; USG: ultrasonography)

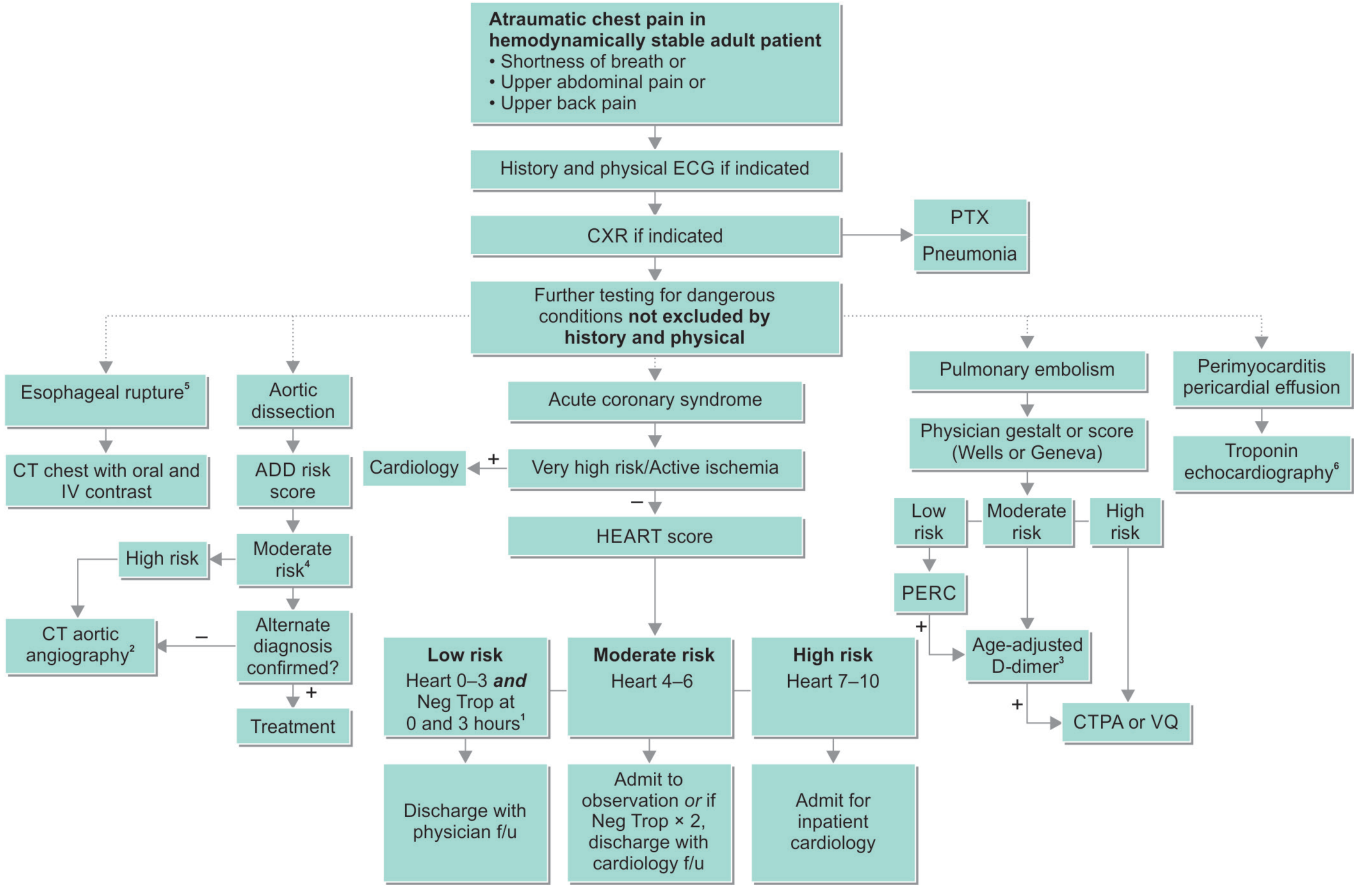
TABLE 6: Causes of chest pain identifiable by ICU screening USG.

System	*Cause*	*Corroborative findings*
Cardiac	• Acute coronary syndrome • Cardiac tamponade • Pulmonary embolism • Aortic dissection	• New regional wall movement abnormality, MR • Pericardial effusion with RA/RV diastolic collapse • RA enlargement, RV strain/dysfunction • Dissection flap with true and false lumen
Respiratory	• Pneumothorax • Pneumonia • Empyema	• Lung point, absent pleural sliding • Shred sign, dynamic air bronchogram • Complex pleural effusion
Upper abdomen	Liver abscess, subpleural collection	Hypoechoic to complex collection

(ICU: intensive care unit; MR: mitral regurgitation; RA: right atrial; RV: right ventricular; USG: ultrasonography)

the sensitivity and specificity of a clinical diagnosis. We cannot deny any longer that such adjuvants as ICU screening ultrasonography (USG)/echocardiography, electrocardiogram (ECG), and arterial blood gas (ABG) analysis have already become an inherent part of clinical examination. These have enabled the identification of life-threatening causes of acute chest pain (the "Big Six"—**Box 1**) with reasonable certainty and speed. We cannot belittle the importance of screening USG in the evaluation of a patient presenting with chest pain, given its ever-expanding scope of application. Some of the common etiologies of chest pain that can be identified with reasonable specificity using USG are listed in **Table 6**. An elaborate discussion of the utility of each of the adjuvant modalities in arriving at the diagnosis will be beyond the scope of this chapter.

Flowchart 2: A model algorithm for evaluation of chest pain.



Atraumatic chest pain in hemodynamically stable adult patient
• Shortness of breath or
• Upper abdominal pain or
• Upper back pain
History and physical ECG if indicated
CXR if indicated
PTX
Pneumonia
Further testing for dangerous conditions **not excluded by history and physical**
Esophageal rupture[5]
CT chest with oral and IV contrast
Aortic dissection
ADD risk score
Moderate risk[4]
High risk
CT aortic angiography[2]
–
Alternate diagnosis confirmed?
+
Treatment
Acute coronary syndrome
Very high risk/Active ischemia
+
Cardiology
–
HEART score
Low risk
Heart 0–3 ***and*** Neg Trop at 0 and 3 hours[1]
Discharge with physician f/u
Moderate risk
Heart 4–6
Admit to observation *or* if Neg Trop × 2, discharge with cardiology f/u
High risk
Heart 7–10
Admit for inpatient cardiology
Pulmonary embolism
Physician gestalt or score (Wells or Geneva)
Low risk
Moderate risk
High risk
PERC
+
Age-adjusted D-dimer[3]
+
CTPA or VQ
Perimyocarditis pericardial effusion
Troponin echocardiography[6]

<table>
<tr><th colspan="3"><i>Modified AHA
Aortic Dissection Detection Risk Score</i></th></tr>
<tr><td><i>High-risk conditions</i></td><td><i>High-risk pain features</i></td><td><i>High-risk examination features</i></td></tr>
<tr><td>• Marfan syndrome
• Connective tissue disease
• Family history of aortic disease
• Known aortic valve disease
• Recent aortic manipulation
• Known thoracic aortic aneurysm</td><td>Chest, back, or abdominal pain described as abrupt in onset, severe in intensity and ripping, tearing, sharp, or stabbing in quality</td><td>• Pulse deficit
• Systolic BP differential
• Focal neurologic deficit
• Hypotension/shock state
• Murmur of aortic insufficiency</td></tr>
<tr><td colspan="3"><i>Low risk:</i> No high-risk features, reassuring CXR
<i>Intermediate risk:</i> 1 high-risk feature or widened mediastinum on CXR
<i>High risk:</i> 2 or more high-risk features</td></tr>
</table>

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Notes:

1. Single troponin may be considered if symptoms >6 hours
2. TEE and MM can be used to exclude aortic dissection in place of CT aorta
3. For assays with cutoff of 500 FEUs, age-adjusted is age x 10; for assays with cutoff of 250 DDUs, age adjustment is age x 5.
4. Patients who are low risk for aortic detection by aortic dissection detection (ADD) score but have widened mediastinum on CXR or other clinical features that concern the clinician should be considered for advanced imaging.
5. Patients with profuse vomiting preceding chest pain, esophageal disease, or recent esophageal instrumentation should be considered for advanced chest imaging to exclude esophageal rupture.
6. Patients with unexplained hypotension should have pericardia effusion excluded by sonography.
7. Known atherosclerotic disease (CAD, PAD, and CVA) garners 2 points, if no known disease, calculate score according to vascular risk factors.

Modified HEART Score		
Patient assessment: *History:* Mostly high-risk symptoms 2 Mix of high- and low-risk symptoms 1 Mostly low-risk symptoms 0 *ECG:* New ischemic change 2 Nonspecific change 1 Normal 0 *Age:* ≥65 years 2 45–64 years 1 <45 years 0 *Vascular disease-maximum 2 points:*[7] Known CAD or 2 Prior stroke or Peripheral artery disease *If no known vascular disease:* ≥3 Vascular risk factors 2 1–2 Vascular risk factors 1 0 Vascular risk factors 0 *Troponin:* >0.12 ng/mL 2 .041–0.120 ng/mL 1 0–0.040 ng/mL 0	*High-risk symptoms:* • Middle or left-sided pain • Heavy chest pain • Diaphoresis • Symptoms relieved by nitrates • Nausea or vomiting • Radiation of pain • Exertional pain *ECG new ischemic changes:* • Ischemic ST depression • New ischemic T wave *Vascular risk factors:* • Obesity (BMI ≥ 30 kg/m^2) • Smoker within 90 days • Diabetes currently being treated • First degree relative with ACS < age 55 years • Diagnosed or treated hypertension • Hypercholesterolemia	*Low-risk symptoms:* • Well-localized pain • Sharp pain • Nonexertional • No diaphoresis • No nausea or vomiting *ECG nonspecific changes:* • Repolarization abnormalities • Nonspecific T-wave changes • Bundle branch blocks • Digoxin effect • Pacemaker rhythms • LVH • Early repolarization • Nonspecific ST changes

PERC score	*Wells score*		*Geneva score*	
• Age ≥50 years	Signs/symptoms of DVT	3	Age >66 years	1
• HR ≥100 beats/min	PE is #1 Dx or equally likely	3	Previous DVT/PE	3
• SpO$_2$ on room air ≤94%	Heart rate >100 beats/min	1.5	Surgery/lower limb fracture in past month	2
• Unilateral leg swelling	Immobilization ≥3 days or surgery within 4 weeks	1.5	Active malignancy	2
• Hemoptysis	Previous DVT/PE	1.5	Unilateral lower limb pain	3
• Recent surgery or trauma	Hemoptysis	1	Hemoptysis	2
• Prior PE/DVT	Malignancy within 6 months	1	HR 75–94 beats/min	3
• Exogenous hormone use	*Low risk*	<2	HR >94 beats/min	5
If one or more criteria is present, patient is PERC positive. If none of these 7 elements are present and clinician's pretest probability is <15%, patent is PERC-negative; no need for further workup, as <2% chance of pulmonary embolism	*Intermediate risk*	2–6	Pain on lower limb deep venous palpation + unilateral edema	4
	High risk	>6	*Low risk*	<4
			Intermediate risk	4–10
			High risk	>10

(ACS: acute coronary syndrome; AHA: American Heart Association; BP: blood pressure; BMI: body mass index; CAD: coronary artery disease; CVA: cerebrovascular accident; CT: computed tomography; CTPA: CT pulmonary angiogram; CXR: chest X-ray; DDU: D-dimer units; DVT: deep vein thrombosis; Dx: diagnosis; ECG: electrocardiogram; FEU: fibrinogen equivalent unit; f/u: follow-up; IV: intravenous; LVH: left ventricular hypertrophy; MRI: magnetic resonance imaging; PE: pulmonary embolism; PAD: peripheral artery disease; PERC: pulmonary embolism rule-out criteria; PTX: pneumothorax; TEE: transesophageal echocardiography)

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SUMMARY

Chest pain is a complex conundrum and it may often not be possible to simplify its evaluation algorithm. It requires great understanding to demystify the symptoms and signs and put them in place with the examination findings. A typical outpatient evaluation may provide us all the time for a detailed and systematic examination; however, the ICU bedside spares no moment. This is further compounded by the fact that the eye only sees what the mind knows! The routine practice of a systematic examination along traditional lines in a nonemergency situation will enhance the skills of clinical examination in a crisis. A model algorithm agreed upon by a Hospital Insurance Company Inc. and available at emupdates.com is shown in **Flowchart 2**. It needs to be re-emphasized that a successful treatment starts with a careful history, examination, and a working diagnosis. The failure to arrive at a working diagnosis and mere symptom-guided approach will defy the art and science behind the practice of clinical medicine.

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Chapter

24

Examination of a Postcardiac Arrest Survivor

Anirban Hom Choudhuri, Sakshi Duggal

ABSTRACT

The examination of a patient following cardiac arrest should be very brief and diligent. The basic life support (BLS) and advanced cardiac life support (ACLS) protocols are highly effective for the return of spontaneous circulation (ROSC) if initiated timely and performed meticulously. Once spontaneous circulation returns, the significance of clinical examination pertains more to prognostication of outcome. The clinical examination includes thorough examination of nervous and locomotor system apart from cardiac and respiratory system. The importance of the other systems varies from individual to individual depending on the cause of cardiac arrest. The period of recovery is also tumultuous and characterized by waxing and waning of signs and symptoms and appearance of certain obscure manifestations which are at times difficult to comprehend.

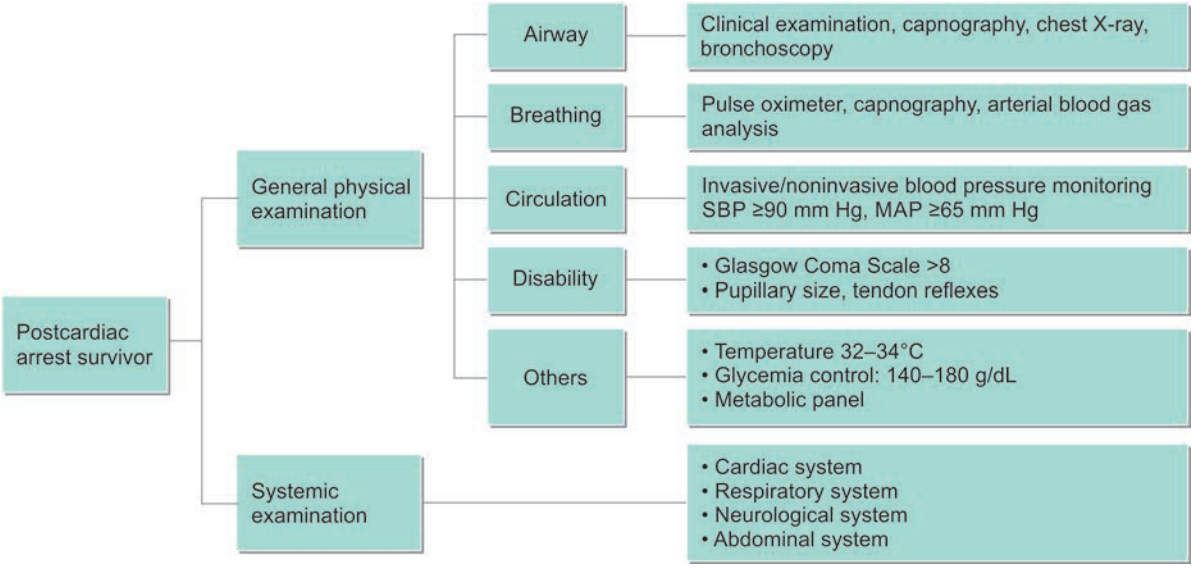
CASE SCENARIO

An 83-year-old female patient was shifted to the intensive care unit (ICU) with a history of fall 15 days back, leading to right-sided fracture of femur shaft. She was managed conservatively with traction and oral medications and was administered low-molecular-weight heparin (LMWH) for thromboprophylaxis. However, during the course of her illness, she suddenly developed breathlessness for a brief period followed by loss of consciousness, circulatory collapse, and asystole. Cardiopulmonary resuscitation (CPR) was immediately initiated and she was resuscitated with return of spontaneous circulation within 40 minutes of cardiac arrest. Computed tomography (CT) pulmonary angiography revealed a massive pulmonary embolism (PE). Upon neurological examination the Glasgow Coma Scale (GCS) score was 5, pupils were reactive to light bilaterally and corneal reflex (CR) was present. The patient was intubated and mechanically ventilated after which she became hemodynamically stable. The prognostication of her outcome is based upon clinical examination to assess improvement in her neurological status along with the detection of any abnormalities in the other systems.

INTRODUCTION

Return of spontaneous circulation (ROSC) is the end point of successful CPR after cardiac arrest. It can be defined as the resumption of perfusion and sustenance of cardiac activity along with significant respiratory effort. Objectively, it is sustained if the chest compressions are not required for 20 minutes and signs of circulation persist (if extracorporeal circulatory support is applied) after CPR. The signs of ROSC include breathing (not just occasional gasp), coughing, or movement along with a palpable pulse or measurable blood pressure during CPR. However, sometimes the expressions of these signs are late (Lazarus phenomenon) and they manifest much after the resuscitation efforts are terminated. Therefore, monitoring of the vital parameters is necessary and recommended for at least 10 minutes after cessation of all resuscitative efforts. Although the return of circulation is a favorable sign its presence alone does not indicate a favorable outcome in postcardiac arrest survivors. Many deaths occur during the first 24 hours after cardiac arrest due to multiorgan dysfunction, which is referred to as postcardiac arrest syndrome and augurs an array of multiorgan involvement due to ischemia and reperfusion. In majority, brain injury and cardiac instability become the major determinants of mortality. Therefore, a comprehensive examination to rule out brain injury and cardiac instability and initiate definitive care becomes vital. And despite being comprehensive and detailed, such examination ought to be quick to guide timely and

Flowchart 1: Examination of a postcardiac arrest survivor: A preview.


Postcardiac arrest survivor
General physical examination
Airway
Clinical examination, capnography, chest X-ray, bronchoscopy
Breathing
Pulse oximeter, capnography, arterial blood gas analysis
Circulation
Invasive/noninvasive blood pressure monitoring SBP ≥90 mm Hg, MAP ≥65 mm Hg
Disability
• Glasgow Coma Scale >8
• Pupillary size, tendon reflexes
Others
• Temperature 32–34°C
• Glycemia control: 140–180 g/dL
• Metabolic panel
Systemic examination
• Cardiac system
• Respiratory system
• Neurological system
• Abdominal system

(SBP: systolic blood pressure; MAP: mean arterial pressure)

targeted care to the victims. In the course of the clinical examination, an attempt should be made to identify the cause of arrest if unrecognized earlier. Once the cause of arrest is identified and targeted management is instituted, the physician should look for confounders that may hinder the postarrest care. These include identification and correction of 5H [hypoxia, hydrogen ion (acidosis of any etiology), hyper-/hypokalemia, moderate to severe hypothermia] and 5T [toxins, tamponade (cardiac), tension pneumothorax, and thrombosis of the coronary or pulmonary vasculature] and are helpful in preventing any prognostication bias. In this chapter, we have outlined a stepwise examination approach for the postcardiac arrest survivors **(Flowchart 1)**.

GENERAL PHYSICAL EXAMINATION

Postarrest examination should begin immediately after ROSC, even before the patient's transportation to the healthcare facility [in case of out-of-hospital cardiac arrest (OHCA) or shift to ICU (in case of an in-hospital cardiac arrest (IHCA)]. Otherwise, discrete systemic management should kick-start immediately upon receipt of the patient in the ICU. This entails a proper head-to-toe examination with an eye on vitals, cardiopulmonary, and neurological status. A preliminary physical examination focusing on ABCDE, i.e., airway, breathing, circulation, and disability, followed by a detailed systematic evaluation should ensue. A brief history can be sought from the accomplices in OHCA or the health personnel in the case of IHCA. Many patients have a history of new-onset or recurrent chest pain, palpitations, breathlessness, light-headedness, and abdominal pain. Many may have symptoms of recent infections, loss of postural tone, focal neurological deficits, seizures, and bleeding. These symptoms can also help us to identify the cause of cardiac arrest and guide appropriate management.

Airway and Breathing

Securing a definitive airway or confirming the correct position of the existing airway is the first crucial step in the examination of the airway. Patients admitted to the ICU may have an endotracheal tube in situ (as in IHCA) or supraglottic airway devices (as in OHCA). Their presence in the correct position can be judged by clinical examination and capnography and confirmed by chest X-ray or bronchoscopy. Once confirmed, the adequacy of oxygenation and ventilation should be checked with pulse oximetry and waveform capnography. If mechanical ventilation is initiated for the first time, it is good to adopt a lung protective strategy and start at a low tidal volume of 6–8 mL/kg and rate of 10–12 breaths/min to target an arterial oxygen saturation ($\mathrm{SpO_2}$) of ≥94% at the lowest possible inspired oxygen concentration ($\mathrm{FiO_2}$) or an end-tidal carbon dioxide concentration ($\mathrm{EtCO_2}$) of 35–40 mm Hg or an arterial $\mathrm{CO_2}$ ($\mathrm{PaCO_2}$) of 40–45 mm Hg.

Circulation

The presence of a palpable pulse and a measurable blood pressure usually reliably indicates successful resuscitation. Continuous blood pressure monitoring through invasive arterial catheters is preferable over noninvasive ones to allow observation of beat-to-beat variation, facilitate better inotropic titration, and ease arterial blood gas analysis. A minimum systolic blood pressure of ≥90 mm Hg or a mean arterial pressure of ≥65 mm Hg is often considered optimum for the maintenance of cardiac output and adequate peripheral perfusion. Circulatory support can be provided in the form of intravenous fluids and vasoactive drugs like epinephrine 0.1–0.5 µg/kg/min, norepinephrine 0.1–0.5 µg/kg/min, or dopamine 5–10 µg/kg/min to maintain hemodynamic stability. Inotropic agents like dobutamine or inodilators like milrinone are occasionally necessary. Measurement of hourly urine output can detect the adequacy of peripheral perfusion. A minimum urine output of ≥0.5 mL/kg/h is an indirect indicator of adequate organ perfusion and hemodynamic stability. Monitoring of hourly urine output with serial estimation of serum creatinine level can help in early detection of acute kidney injury (AKI) and timely initiation of renal replacement therapy (RRT).

Disability

Neurological disability is a major determinant of postcardiac arrest survival. Early determination of neurological damage is often impossible up to 72 hours of postarrest and also inaccurate. However, early hemodynamic optimization should be attempted for good neurological outcomes even if neurological damage is indeterminable. The consciousness should be assessed early after resuscitation and reassessed at frequent intervals. A GCS score of 8 or less indicates neurological injury postresuscitation and entails securing a definitive airway if not already done during resuscitation. Pupillary size and superficial and deep tendon reflexes should be examined to determine the site of the neurological lesion and guide prognosis (discussed later under the heading of neurological examination). Targeted temperature management and glycemic control are important contributors in preventing further neurological damage.

- *Temperature:* Targeted temperature management is an essential component of postcardiac arrest care. The patient's core temperature should be continuously monitored if the patient is planned for therapeutic hypothermia, preferably with an esophageal thermometer, or through a bladder catheter in nonanuric patients, or through a pulmonary artery catheter. Axillary or oral temperature monitoring, even though convenient is less reliable. Tympanic membrane probes are both unreliable and unavailable in many places. Comatose adult patients with ROSC after OHCA cardiac arrest and ventricular fibrillation, pulseless electric activity or asystole or an IHCA of any initial rhythm (Class IIb, LOE B) should be cooled to 32–34°C (89.6–93.2°F) for at least 12–24 hours. The beneficial effect of hypothermia can be seen if these temperatures are achieved within 10–20 minutes of ROSC but are often lost once if delayed. This highlights the importance of early temperature management in achieving good neurological outcomes. A rise in temperature to >37.7°C should be actively avoided for at least 72 hours after ROSC in comatose patients.
- *Glycemic control:* Euglycemia should be maintained for good neurological recovery. Moderate glycemic control with blood sugars between 144 and 180 mg/dL should be targeted in patients after ROSC. Strict glycemic controls with blood glucose between 80 and 110 mg/dL may result in recurrent hypoglycemic episodes that may exaggerate the neurological damage.

Investigations: Metabolic Panel

A basic metabolic panel with arterial blood gas analysis should be done in the early stages of management. A complete blood count, serum electrolyte levels, blood lactate levels, and arterial blood gases are a few initial investigations that help delineate the severity of the underlying condition. The presence of severe anemia, infection, hypo-/hyperkalemia, and metabolic acidosis can guide remedial measures. Assessment of acidosis, hypoxia, or hypercapnia on blood gas analysis can further help to find the cause of arrest and gather feedback on ventilatory status. Normal or decreasing serum lactate level is a favorable sign, which signifies improving peripheral perfusion and hemodynamic stability. Toxicology testing can be considered in patients with a suggestive history and where treatment can be modified according to the findings.

RESPIRATORY EXAMINATION

Pulmonary changes can cause or predispose to cardiac arrest. Hypoxia, tension pneumothorax, and pulmonary thromboembolism are some of the major pulmonary causes of cardiac arrest. Pulmonary dysfunction can also occur after cardiac arrest. Hydrostatic pulmonary edema from left ventricular dysfunction, noncardiogenic edema from inflammatory, infective, severe pulmonary atelectasis, physical injuries, or aspiration can occur after ROSC. Therefore, a thorough examination of respiratory system along with radiological investigations and blood gas analysis are necessary to assess the adequacy and status of oxygenation and ventilation.

Inspection

Respiratory system examination starts with airway inspection. The need for airway management and mechanical ventilation is assessed with regard to the cause of arrest and the patient's antecedent consciousness level and hemodynamic status. Patients whose airway is not secured with endotracheal intubation should be intubated first and the tube position is confirmed clinically through capnography or chest X-ray. This is followed by inspection of chest shape, symmetry fullness or depression, and bilateral movement. The fullness of chest on one side with asymmetrical chest expansion may suggest endobronchial intubation, pleural effusion, or pneumothorax. Observation of respiratory rate and pattern, presence of spontaneous efforts, and patient–ventilator asynchrony should be examined and corrected. Suprasternal or intercostal recession with use of accessory respiratory muscles may suggest air hunger in a mechanically ventilated patient. The presence of bruises may suggest physical injuries due to sternal or rib fractures in the case of trauma patients or during resuscitation. The presence of needle puncture in the second intercostal space suggests tension pneumothorax as the cause of respiratory arrest. Any audible stridor or wheeze should also be noted.

Palpation

Surface temperature should be assessed during palpation. Position of the trachea, apex beat, and symmetrical expansion of the chest by a measuring tape should be done. Tracheal deviation to the opposite side might be present in the case of pneumothorax or massive pleural effusion. Diminished chest wall movements in case of pneumothorax or pleural effusion, or increased expansion in ipsilateral endobronchial tube position might be present. Any palpable accompaniments like friction fremitus, and palpable coarse crepitations should be seen. Subcutaneous emphysema might be evident as a feature of rib fracture postresuscitation and the presence of a bronchopleural fistula at times.

Percussion

Conventional percussion over the lung fields should be done. Stony dullness in case of pleural effusion or hemothorax or tympanic percussion in pneumothorax should be observed. It is also important to delineate hepatic and cardiac dullness on percussion. Obliteration of liver dullness with the upper border of dullness at the sixth intercostal space might suggest right-sided pneumothorax on percussion.

Auscultation

Auscultation of breath sounds and adventitious sounds (rhonchi, crepitations, and pleural rub) should be noted. Diminished breath sound might suggest ipsilateral pleural effusion or pneumothorax. Presence of crepitations might point toward pulmonary edema or an infective pathology or aspiration.

Arterial Blood Gas Analysis

Blood gas evaluation is crucial to assess the adequacy of oxygenation or ventilation in a mechanically ventilated patient. The intensivists should target an SpO_2 of >94% on the minimum FiO_2. Ventilation should be initiated at a rate of 10–12 breaths/min and a tidal volume of 6–8 mL/kg to attain an $EtCO_2$ of 35–40 mm Hg, or a $PaCO_2$ of 40–45 mm Hg.

Patients often develop regional ventilation–perfusion mismatch due to pulmonary edema, regional atelectasis, and hypotension contributing to decreased arterial oxygen content. The severity of this pulmonary dysfunction can be measured through arterial to inspired oxygen concentration (PaO_2/FiO_2) ratio. A PaO_2/FiO_2 ratio of <300 mm Hg is suggestive of acute lung injury. This along with acute onset of bilateral infiltrates on chest X-ray and a pulmonary artery pressure <18 mm Hg or no evidence of left atrial hypertension leads to the diagnosis of acute respiratory distress syndrome (ARDS). A PaO_2/FiO_2 ratio <300 or >200 mm Hg separates acute lung injury from ARDS. A protocol of low volume high rate lung-protective ventilation with use of positive end-expiratory pressure (PEEP) and titrated FiO_2 strategies can improve pulmonary function.

Radiological Investigations

- *Chest X-ray:* Early verification of correct positioning of the endotracheal tube and an evaluation of lung parenchyma are amongst the initial and most important diagnostic advantages of a bedside chest X-ray. It also helps identify complications from chest compressions (e.g., rib fracture, pneumothorax, and pleural effusions) or evaluation of other lung pathologies (e.g., pulmonary edema and pneumonia).
- *Computed tomography:* A contrast chest CT is helpful in identification of a suspected PE or aortic dissection.
- *Ultrasonography:* Use of bedside sonography is another fast modality for urgent evaluation of patient. It can be used to confirm the correct tube placement or presence of pneumothorax, pleural effusion, thoracic or abdominal bleeding, or volume status according to the inferior vena cava collapsibility index. Pericardial tamponade and structural heart diseases such as hypertrophic cardiomyopathy can also be identified rapidly through bedside echocardiography. In patients with suspected PE who cannot undergo CT, sonographic evidence of right ventricular dysfunction or direct visualization of clot can confirm the diagnosis.

CARDIAC EXAMINATION

Cardiovascular diseases leading to coronary ischemia are one of the most important causes of cardiac arrest. Cardiac arrest and resuscitation further compromise the effective functioning of the heart to maintain the hemodynamic stability and peripheral or coronary perfusion. Thus, a through cardiac evaluation is essential in a resuscitated patient to optimize postarrest recovery.

It should focus on identifying dysrhythmias, adequacy of circulation assessed by blood pressure and pulse strength in all the limbs, or presence of cardiac murmurs if any. Low intensity or distant heart sounds should prompt consideration for underlying pericardial tamponade.

Cardiovascular examination starts right form general physical examination with assessment of hemodynamics. A quick assessment of peripheral pulses for rate, rhythm, volume, and any special character and examination of neck veins for engorgement or collapsibility with pulsation or hepatojugular reflex if present should be done. Invasive arterial blood pressure monitoring should be done continuously to assess the adequacy of systematic, coronary, and cerebral perfusion. Fluid administration and inotropic titration to maintain a minimum systolic blood pressure of ≥90 mm Hg or a mean arterial pressure of ≥65 mm Hg should be done.

Inspection

Inspection of precordium for any deformity, engorged veins or pulsation or the cardiac areas for any visible pulsations should be done. Bruising over the precordium or sternum might point toward sternal or rib fracture as a result of trauma or aggressive chest compressions.

Palpation

All four cardiac areas must be palpated for evidence of pulsation, palpable heart sound, or thrill. Apex beat should be localized at the mitral area while a parasternal heave should be elicited at the tricuspid area. The four cardiac areas are as follows:

1. *Mitral area:* Left fifth intercostals space, ½” medial to the mid-clavicular line
2. *Aortic area:* Right second intercostal space (close to sternum)
3. *Pulmonary area:* Left second intercostal space (close to sternum)
4. *Tricuspid area:* Left forth intercostal space (close to sternum).

Auscultation

The heart areas should be auscultated for cardiac sounds, new-onset murmurs, or any adventitious sounds opening snap, ejection click, S3, and S4. Auscultation over carotid areas should also be done to assess for presence of bruit.

12-lead Electrocardiogram

A 12-lead electrocardiogram (ECG) should be obtained as soon as possible in a patient postcardiac arrest, presence of ST-segment deviation, T-wave abnormalities, dysrhythmias, new-onset conduction defects, QT-interval, and presence of low voltage (suggestive of tamponade). Initial and repeat ECG should be compared for early detection of ongoing or advancing myocardial ischemia. A 64-lead continuous ST mapping using a chest vest may be done where indicated as it has higher sensitivity and specificity compared to a standard ECG.

Echocardiography and Ultrasonography

Bedside echocardiography within the first 24 hours after arrest helps in early assessment of myocardial function and serves as a useful adjunct to basic modalities, especially if there is suspicion of underlying cardiac ischemia and a nondiagnostic initial ECG. Global hypokinesia is a common finding postcardiac arrest, but presence of regional wall motion abnormalities suggests cardiac ischemia. New-onset regurgitant murmurs and evidence of pericardial tamponade can also be evaluated bedside sonographically.

Coronary Angiography

Coronary angiography should be undertaken if there is clinical, ECG, or sonographic evidence of myocardial ischemia. This should be followed by CT pulmonary angiography if coronary angiography fails to identify causative lesions.

Blood Investigations

Baseline and serial measurements of cardiac biomarkers (troponin and CPK-MB) may be obtained as needed to direct initial management of patients' post-ROSC.

NEUROLOGICAL EXAMINATION

Early neurological examination and outcome prognostication is a crucial part of postarrest care. This is especially important when decisions to continue or withdraw life support have to be made. Neuroprognostication should be taken as a multimodal approach as no single test has sufficient specificity to eliminate false positives. Accurate and reliable tools with a false-positive rate (FPR) approaching 0% are needed to foresee a poor outcome, defined as death, persistent unresponsiveness, or the inability to undertake independent activities after 6 months. These neurological parameters have to be assessed postresuscitation as no prearrest or intraarrest parameters (duration of arrest, witnessed arrest, bystander resuscitation, or presenting rhythm) alone or in combination can accurately predict outcome in resuscitated patients. However, no clinical neurological signs reliably predict poor outcome in <24 hours after cardiac arrest. This assessment should be done in the absence of confounding factors (such as hypotension, seizures, sedatives, or neuromuscular blockers) to get accurate results.

Clinical Examination

Assessment of consciousness is one of the first steps in general physical examination of the patient. It helps to decide for need for intubation but helps in neuroprognostication too. A score of <5 on Glasgow Coma Scale-Motor scale (GCS-M) and normal brain stem reflexes in patients undergoing therapeutic hypothermia are associated with better prognosis. While a GCS-M score of 1–2 with absent bilateral pupillary reflex and corneal are known to have a poor outcome.

Assessment of ocular reflexes gives a quick and reliable bedside assessment of patient prognosis. Absence of both pupillary light and CRs after 72 hours of cardiac arrest predicts poor outcome in postcardiac arrest patient with high reliability. The absence of vestibulo-ocular reflexes at >24 hours (FPR 0%) or GCS score <5 at more than 72 hours after arrest (FPR 0%) are lesser reliable parameters for predicting poor outcome. Assessment of myoclonus is not recommended for neuroprognostication.

Ocular reflexes: Ocular reflexes currently used for neurological prognostication after cardiac arrest include pupillary reflex and CR. It is recommended that both reflexes should be absent simultaneously at the time of assessment for reliable prediction of neurological outcome. However, confounding factors like sedation or neuromuscular blockade should be absent during evaluation.

Pupillary light reflex (PLR) is the temporary reduction of pupil size by a light stimulus. And absence of bilateral PLR in patients 72 hours after cardiac arrest in a comatose patient suggest poor outcome. Quantitative evaluation of PLR using portable pupillometers (automated pupillometry) provides an objective and quantifiable measurement of the pupillary response, thus reducing the interobserver variability. It is measured as a percentage reduction of pupillary size. Calculation of Neurological pupil index (NPi) further increases the sensitivity of the test as NPi can predict unfavorable outcome with no false positive results from 24 hours to up to 72 hours from ROSC. It includes dynamic parameters of the pupillary response (including constriction and dilation velocity, size and percentage size reduction after stimulation) using a proprietary algorithm and a value ≥3 is considered normal.

Corneal reflex is in an ocular reflex, characterized by blinking of the eye in response to stimulation of the outer margin (limbus) of the cornea with a cotton wisp, water squirt, or even air. Bilateral absence of this reflex at 72 hours or later after ROSC suggests a poor neurological outcome in postarrest comatose adults.

Electroencephalography

Electroencephalography (EEG) is a useful tool for neuroprognostication in a patient postcardiac arrest. The EEG background continuity is a most important prognostic marker for outcome and can be categorized as continuous, discontinuous, burst suppression (50–99% suppression periods), or suppression (>99% activity, <10 μV amplitude). Presence of malignant EEG patterns, defined as suppressed background with or without periodic discharges and burst-suppression after 24 hours of cardiac arrest, is associated with poor prognosis. In patients undergoing therapeutic hypothermia, a reactive or a continuous EEG background after rewarming suggests a favorable prognosis while burst suppression pattern, electrocerebral inactivity, and status epilepticus are associated with poor outcome. Continuous EEG monitoring can also be done in patients undergoing therapeutic hypothermia for early detection of seizures and to help differentiate convulsions from hypothermia-induced shivering. It is suggested that EEG evaluation to decipher the prognosis should be done after the end of therapeutic hypothermia and after sedation has been cleared.

Somatosensory-evoked potentials (SSEP) can also be helpful as its results are not affected by sedation and can be done in patients undergoing therapeutic hypothermia. Bilateral absence of N20 cortical response to median nerve stimulation on SSEP2545 hours after the arrest is said to be associated with poor prognosis.

Even though the presence of myoclonus or status myoclonus within 96 hours after ROSC does not reliably predict a poor neurological outcome and is not recommended; its assessment in combination with other tests may be used for neuroprognostication.

Radiological Imaging

Computed tomography imaging and magnetic resonance imaging (MRI) are the most commonly studied modalities for neuroprognostication. CT studies can provide a qualitative description of brain structures like structural lesions, stroke, or intracranial hemorrhage that may have contributed to cardiac arrest as well as a quantitative description of grey matter: White matter Hounsfield unit ratio to assess the prognosis. Presence of diffuse and extensive anoxic injury, extensive cortical and subcortical lesions, or cerebral edema on brain imaging suggests a poor outcome prognosis. Other neuroimaging modalities like single-photon emission CT, cerebral angiography, and transcranial Doppler are also in evaluation.

Biochemical Markers

Measurement of biomarkers in the blood or cerebrospinal fluid is recently in use as early predictors of poor outcome in comatose cardiac arrest survivors. These biomarkers are released from dying neurons or glial cells in the brain and are unlikely to be confounded by sedation or neuromuscular blockade. Most commonly used biomarker is neuron-specific enolase with a value >60 μg/L measured between 24 and 72 hours of ROSC, which is predictive of a poor neurological outcome. Other markers such as S-100B protein, glial fibrillary acidic protein (GFAP), or Tau protein are unreliable for assessment of neurological outcome.

ABDOMINAL EXAMINATION

A quick abdominal examination should be done to rule out any distension, guarding, or presence of pulsatile masses. Proctoscopy may be done in suspected cases to assess gastrointestinal bleeding. The patient should also be assessed for complications related to CPR. CT of abdomen and pelvis should be considered in patients with suspected abdominal etiology.

SUMMARY

Postcardiac arrest care was carried out for the patient for the next 3 days. Patient was mechanically ventilated with endotracheal intubation and weaned and extubated 3 days after the return of spontaneous circulation. Patient's hemodynamics were maintained within normal limits with the use of fluids and vasopressors. The initial s1Q3T3 changes as seen on ECG were slowly reverted to sinus rhythm and therapeutic thrombolysis using unfractionated heparin. The temperature was maintained between 34 and 36°C during the first 24 hours postarrest. Glycemic control was also ensured with 100–140 g/dL. Chest X-ray showed oligemia initially which ultimately cleared after initiation of therapeutic thrombolysis. Other systems did not reveal any significant findings. Upon full recovery, patient was fully conscious and awake with GCS 25/25, hemodynamically stable and maintaining saturation on room air. She was finally discharged on day 8 of arrest with conservative management of the fractured femur.

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Chapter

25

Examination of a Patient for Difficult Airway in the Intensive Care Unit

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ABSTRACT

Airway management in the intensive care unit (ICU) is different compared to the operating room and is considered challenging due to a number of factors, which include patient-related, environment-related, and operator-related factors. Critically ill patients due to their severe physiologic derangements are at increased risk of cardiovascular collapse and death during tracheal intubation hence called the physiologically difficult airway. Airway assessment of critically ill patients is difficult due to a multitude of reasons such as uncooperative patients, emergent need of intubation, sedation, and delirium. No particular test is sensitive or specific enough to detect difficult airways among critically ill patients. Thyromental distance, upper lip bite test, sternomental distance (SMD), and Mallampati test are few tests that are found to be beneficial for airway assessment in ICU. Some scoring systems such as LEMON and Wilson score which incorporate some measurements and examination for prediction of anatomically difficult airway can be used. Imaging like X-ray and computed tomography (CT) scans can be used for radiological assessment of a few masses and systemic diseases that can be associated with anatomically difficult airway. Physiologically difficult airway like hypoxemia, hypotension, metabolic acidosis, right ventricular (RV) failure, neurological derangement, and gut derangement should be recognized in critically ill patients as measures should be taken to reduce complications associated with endotracheal intubation. Few scoring systems which incorporate both anatomically and physiologically difficult airway parameters like MACOCHA scoring and HEAVEN scoring systems can be used as part of airway assessment in ICU. It is important to remember that despite using clinical examination, tests, and scoring systems, critically ill patients are at increased risk of airway-related complications; hence, all intubations should be considered potentially difficult and measures must be taken to reduce complications.

INTRODUCTION

Patients admitted to the intensive care unit (ICU) due to varied reasons may need airway management, including tracheal intubation. Intubation in a critically ill ICU patient is different from intubation in the operating room. Intubation in the ICU is considered a high-risk procedure for various reasons concerning the patient, like hemodynamic instability, hypoxemia, metabolic acidosis, raised intracranial pressure (ICP), and coagulopathy. Several other factors, like the environment of the ICU and the skill of the operators and assistants in the ICU, also have an impact on the procedure. The 4th National Audit Project found that factors that may contribute to poor outcomes regarding airway management are failure to assess and identify potential difficulty, and poor judgment in management planning. They reported that 61% of airway-related events in the ICU were associated with death or permanent neurological damage compared to 14% in the operating room. The incidence of difficult intubation in ICU is about 10% and varies from 5 to 25% as per various studies. In the recent “INTUBE” study—intubation practices among critically ill patients, a multinational, a multicenter observational study found that cardiovascular complications were much higher than respiratory complications and accounted for about 43%. Hence, it is important to realize that airway management in the ICU is completely different from that in the operating room with patients having both anatomically and physiologically difficult airways. Airway assessment in this subset of patients must go beyond carrying out a series of bedside tests and incorporate steps to identify different problems likely to be encountered during each step and devise a reasonable strategy to tackle the same.

CHALLENGES IN THE AIRWAY EXAMINATION OF AN INTENSIVE CARE UNIT PATIENT

Unlike preoperative assessment of patients for elective surgery, airway examination in critically ill patient is often challenging. There are multiple reasons for this, including the need for emergent tracheal intubation in most patients, leaving minimal or no time for airway examination, or the patient being uncooperative or comatose. Nevertheless, every attempt must be made to examine the airway when feasible and have an objective assessment for better preparedness. It is important to remember that none of the tests for assessment of anatomical difficulty in airway management are perfect. Therefore, preparedness for a failed airway at all times is paramount.

Anatomically Difficult Airway

The term anatomically difficult airway is used when there is difficulty in bag-mask ventilation, visualization of glottic opening, or passing the endotracheal tube through the opening.

- *Difficult airway:* When a conventionally trained anesthetist experiences difficulty with tracheal intubation, difficulty with mask ventilation, or both.
- *Difficult mask ventilation:* When the unassisted anesthetist cannot maintain the arterial oxygen saturation (SpO_2) above 90% by mask ventilation using 100% oxygen and positive pressure mask ventilation (provided it was above 90% prior to induction) or cannot reverse signs of inadequate ventilation [e.g., absence of exhaled CO_2, chest movement, and spirometric measures of exhaled gas flow or the presence of cyanosis, tachycardia, and hypertension (HTN)].
- *Difficult laryngoscopy:* When it is not possible to visualize any portion of the vocal cords with conventional laryngoscopy.
- *Difficult endotracheal intubation:* When the insertion of an endotracheal tube takes more than three attempts or longer than 10 minutes to perform using conventional equipment.

CASE SCENARIO

A 65-year-old male a known case of (k/c/o) diabetes mellitus (DM), HTN diagnosed with carcinoma of the hard palate receiving chemotherapy and radiotherapy develops fever, loose motions, and abdominal pain. Patient is shifted to the ICU where patient is drowsy, arousable, has a heart rate of 130 beats/min, blood pressure of 90/60 mm Hg, respiratory rate 30 breaths/min, SpO_2 of 90% at room air, and on auscultation chest is clear. Arterial blood gas shows metabolic acidosis with hyperlactatemia. In view of septic shock, altered sensorium, and increased work of breathing, it was decided to intubate the patient. How will you assess this patient in view of his airway and prepare for intubation?

History and Symptomatology

A detailed history of the patients may not be possible in patients who present to the ICU and need emergency airway management. Patients with challenging anatomy must be identified, like maxillofacial trauma, cervical spine injury, airway injuries, burns, retropharyngeal abscesses, and upper airway cancers. Such patients are classified as having an anticipated difficult anatomical airway, and adequate preparation for airway management should be done. Few chronic diseases such as rheumatoid arthritis, ankylosing spondylitis, and DM can cause limited joint mobility. Rare syndromes such as Pierre Robin, Klippel–Feil, and Treacher Collins may coexist who have difficult airway anatomy. Obese patients with a history of obstructive sleep apnea (OSA) may be at risk for desaturation during intubation, and a history of OSA must be asked. A history of stridor, dysphagia, hoarseness, or voice change may suggest the possibility of a difficult airway and an increased risk of aspiration.

Clinical Examination: The Airway Examination

No airway assessment tool in isolation is sensitive to predict difficuly airway in critically ill patients. Obvious abnormalities like a short neck, reduced mouth opening, receding jaw, and buck teeth can all lead to difficult laryngoscopy. The presence of beard, edentulous, obese patients, and snorers can lead to difficult mask ventilation. Radiation to the neck is also a significant predictor of difficult mask ventilation, as it causes the development of fibrotic, noncompliant tissue affecting the airway.

Mouth opening is an important factor that determines the ability to insert most devices. A direct laryngoscope blade (Macintosh) requires at least 3 cm inter incisor space for blade to be inserted. Few video laryngoscope blades can pass with less than 3 cm mouth opening also.

The thyromental distance, described as the distance from the thyroid notch up to the mental prominence with the head fully extended, is a test done to see mandibular space with distance more than 6.5 cm considered normal. A distance between 6 and 6.5 cm predicts difficult laryngoscopy, but intubation is usually possible. A distance less than 6 cm suggests intubation using conventional, direct laryngoscopy, which may be difficult or impossible. **Figure 1** shows landmarks to measure thyromental distance and its interpretation.

Upper lip bite test can be done as an assessment of jaw mobility. The patient is asked to protrude the lower jaw and bite the upper lip. Higher the grade, higher is the risk of difficult intubation. **Figure 2** shows grading based on upper lip bite test.

Sternomental distance (SMD) is defined as the distance between the mentum and sternum with the head fully extended and the mouth closed. A value of less than 12.5-13.5 cm can predict a higher incidence of difficult intubation. SMD is considered an indicator of head and neck mobility, with head extension playing an important part in obtaining a good laryngeal view with a conventional laryngoscope.

Neck mobility also affects laryngoscopy and intubation. Limited neck mobility makes achieving sniffing position difficult, as well as manipulation of laryngoscope and tube. Laryngeal tumors, vocal cord palsies, and subglottic stenosis can affect tracheal tube sizing or suitability for awake fibroscopy. The presence of stridor, with or without the presence of respiratory distress, can be a clue to underlying pathologies and should alert the airway provider of possible complications.

Some scoring systems, such as Mallampati score, LEMON score, and Wilson score, can be used to predict difficult airway. **Figure 3** shows Mallampati scoring based on structures seen. **Table 1** describes components of LEMON airway assessment score. **Table 2** describes Wilson scoring for difficult intubation.

Imaging

Even though imaging like chest X-ray, computed tomography (CT) scan do not form a part of the routine

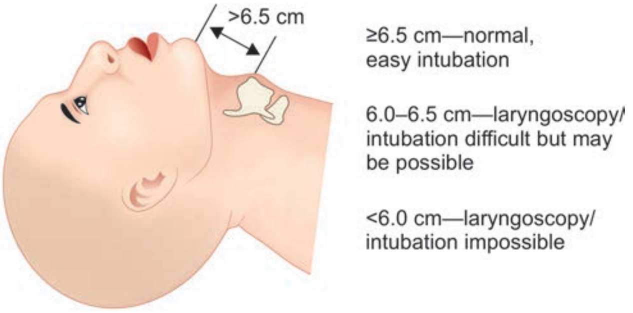
>6.5 cm
≥6.5 cm—normal, easy intubation
6.0–6.5 cm—laryngoscopy/intubation difficult but may be possible
<6.0 cm—laryngoscopy/intubation impossible

Fig. 1: Thyromental distance.

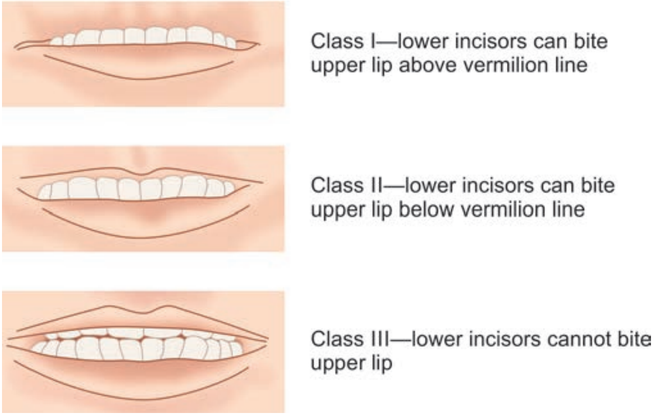
Class I—lower incisors can bite upper lip above vermilion line
Class II—lower incisors can bite upper lip below vermilion line
Class III—lower incisors cannot bite upper lip

Fig. 2: Upper lip bite test.

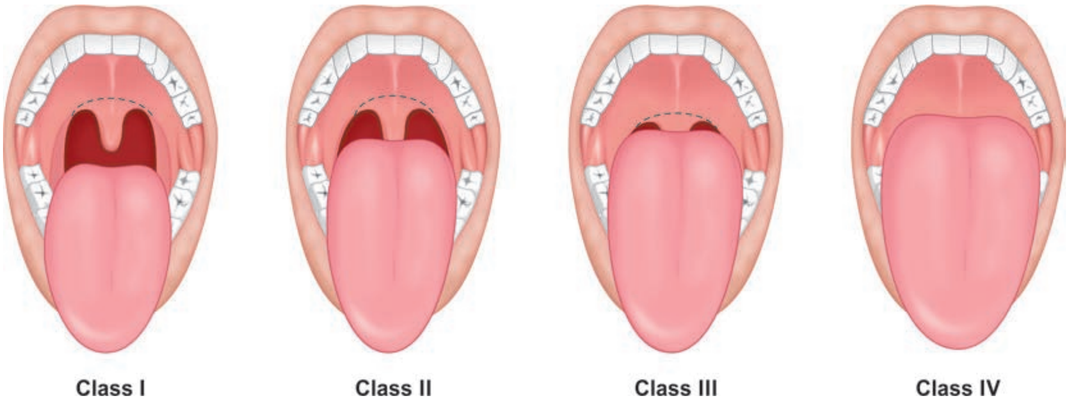
Class I
Class II
Class III
Class IV

Fig. 3: Mallampati scoring—Class 1 to 4.

TABLE 1: LEMON airway assessment.

L	Look externally (facial trauma, large incisors, beard or moustache, large tongue)
E	Evaluate 3-3-2 score: • Interincisor distance—3 finger breadth • Hyomental distance—3 finger breadth • Thyroid to mouth distance—2 finger breadth
M	Mallampati score
O	Obstruction like epiglottitis, peritonsillar abscess, and trauma
N	Limited neck mobility

TABLE 2: Wilson difficult intubation score.

Parameter	*Levels*	*Score*
1. Weight	<90 kg 90–110 kg >110 kg	0 1 2
2. Head and neck	Above 90°	0
3. Movement	About 90° Below 80°	1 2
4. Jaw movement—interincisor gap (IG) and subluxation (SLux)	IG > 5 cm or SLux > 0 IG < 5 cm or SLux = 0 IG < 5 cm or SLux < 0	0 1 2
5. Receding mandible	Normal Moderate Severe	0 1 2
6. Buck teeth	Normal Moderate Severe	0 1 2

assessment of patients; however, in a few cases they can provide useful information. In patients with neck mass, the retrosternal extension and compression on airway structures and distal obstruction can be made out. Lateral neck X-rays in a few cases, like rheumatoid arthritis and ankylosing spondylitis, can show a reduction in space (5 mm) between the C1 spinous process and the occiput, which is an indicator of difficult intubation. CT and magnetic resonance imaging can be used for reconstruction and understanding of airway structures particularly soft tissue. However, none of these tests are foolproof and intensivists may encounter difficult airway despite normal radiographs, so preparation is necessary. Airway assessment by ultrasound is becoming more popular these days. Detection of the cricothyroid membrane in cases of anticipated difficult airway as a landmark for cricothyroidotomy can be done. Postintubation tracheal placement of an endotracheal tube can be confirmed with ultrasound. Fasting assessment can be done by gastric ultrasound to look for a full stomach.

Physiologically Difficult Airway

The physiologically difficult airway is defined as one in which severe physiologic derangements place patients at increased risk of cardiovascular collapse and death during tracheal intubation and transition to positive pressure ventilation. A critically ill patient with a physiologically difficult airway may present with hypoxemia, hypotension, RV failure, metabolic acidosis, and neurologic injury. Noncritically ill patients with a physiologically difficult airway are those who are obese, pediatric, pregnant, or at risk of aspiration during tracheal intubation (after a meal, with gastroesophageal reflux disease, intestinal obstruction, etc.). Recognition of this high-risk group of patients is essential to implement measures to avoid complications during tracheal intubation. Unlike the anatomically difficult airway, where placing the endotracheal tube safely within the trachea is the primary goal, in patients with a physiologically difficult airway, prevention of adverse events is equally important during airway management.

Some of physiological derangements which predispose to complications are:

- *Hypoxemia:* Critically ill patients who are hypoxemic are at increased risk of cardiopulmonary complications during and after intubation, like desaturation, hypoxic brain injury, cardiac dysrhythmias, and cardiac arrest. Hypoxemic type 1 respiratory failure is a type of respiratory failure wherein there is a failure to maintain adequate arterial oxygenation. Critically ill patients with conditions like pneumonia, pulmonary edema, acute respiratory distress syndrome (ARDS), and asthma present with acute hypoxemic respiratory failure. The mechanisms of hypoxia in these patients are a ventilation–perfusion mismatch as well as a shunt. Shunt fraction in these patients is significantly increased; hence, these patients' hypoxia may not improve just with higher fraction of inspired oxygen (FiO_2). Hence, these patients are at increased risk of cardiopulmonary complications due to the increased risk of rapid desaturation during intubation attempts. In these patients, early identification of potential physiologically difficult airway and the use of measures to prolong safe apnea time prior to intubation become crucial, irrespective of their anatomic airway assessment.

Different methods to prolong safe apnea time are preoxygenation, optimal positioning, and apneic oxygenation. The aim of preoxygenation is to denitrogenate the lungs to improve the oxygen reservoir and, hence, prolong safe apnea time during attempts at intubation. Preoxygenation in the operation theater (OT) for noncritically ill patients is usually done by a tight-fitting mask connected to anesthesia circuit with 100% oxygen for 3–5 minutes, with the patient asked to take normal tidal volume breaths. However, in critically ill patients, this method may not be effective due to shunt physiology. Hence, it is recommended to use noninvasive ventilation (NIV) for preoxygenation, as it recruits collapsed alveoli leading to more effective preoxygenation. High-flow nasal oxygen (HFNO) by cannula can also be used for preoxygenation and has been found to be more effective than face mask and slightly inferior to NIV. The other added advantage of HFNO is that continued oxygen can be provided even during laryngoscopy until endotracheal tube placement.

Optimizing position during preoxygenation and intubation can also prolong safe apnea time. The head-up position or ramped position has been found to be better compared to the supine position and has been recommended to be used in patients with high risk of desaturation, especially obese patients. Another method of maintaining saturation during apnea is to provide apneic oxygenation with the help of standard nasal cannula or HFNO. This works on the principle of mass flow of oxygen because of the pressure generated due to the differential diffusion capacity of O_2 and CO_2. Hence, these critically ill hypoxic patients are at very high risk of adverse events during intubation. Preoxygenation for at least 3–5 minutes with the help of noninvasive positive pressure ventilation with an inspiratory pressure of 5–15 cmH_2O, a positive end-expiratory pressure (PEEP) of 5 cmH_2O and a tidal volume of 6–8 mL/kg in a head-up position or with the help of a high flow nasal cannula with oxygen at 70 L/min is essential. Despite this, patients' apnea time may not increase, and they may rapidly desaturate due to their underlying altered physiology. Hence, in these patients it is imperative to secure the airway in the first attempt itself. In these patients, operators with maximum experience and methods to improve the first pass success rate must be applied.

- *Hypotension:* Patients with preexisting hypotension and shock are at increased risk of cardiopulmonary complications such as bradycardia, cardiac arrest, and mortality postintubation. Not all critically ill patients have overt shock. Some patients, due to compensatory sympathetic mechanisms, may have tachycardia and borderline hypotension. Shock index, defined as heart rate/systolic blood pressure, is an early sign of shock and has been found to correlate with an increased risk of cardiovascular complications if the value is more than 0.8. Common causes of hypotension among critically ill patients are hypovolemia, decreased peripheral vascular resistance, and capillary leaks. If hypovolemia is the cause, adequate fluid resuscitation prior to intubation should be done if the heart function is reasonably good. However, not all patients are fluid responsive and may need vasopressors to maintain blood pressure. Critically ill hypotensive patients on induction of anesthesia and positive pressure ventilation can rapidly decompensate and develop cardiac arrest. Hence, these hypotensive patients need to be assessed to determine if they are adequately volume repleted and vasopressors should be initiated prior to attempting intubation. Drugs used for intubation can also affect the hemodynamics of the patient. Induction agents like etomidate and ketamine have been found to be more hemodynamically stable compared to agents like propofol and thiopentone.
- *Right ventricular failure:* Patients with right ventricular (RV) failure are at increased risk of cardiovascular collapse during intubation and subsequent positive pressure ventilation. A failed right ventricle is unable to meet increased demands that can lead to dilatation, retrograde flow, decreased coronary perfusion, hypotension, and cardiovascular collapse. Positive pressure ventilation can lead to an increase in pulmonary pressure that further increases afterload as well as a reduction in preload. Hypoxia and hypercarbia can increase pulmonary artery pressure and worsen RV failure. If these patients need intubation, careful preparation and management is necessary. During intubation, brief periods of apnea can lead to significant hypoxia and hypercarbia, which can worsen the RV failure. Adequate preoxygenation with NIV, use of HFNO during intubation, and head-up positioning need to be done to avoid hypoxia and hypercarbia during intubation. Fluid loading is also difficult in patients with RV failure as it can lead to a

shift of the septum, leading to decreased left ventricular preload, cardiac output, and shock. Vasopressors like norepinephrine need to be started if associated hypotension, as they increase mean arterial pressure without increasing pulmonary artery pressure.

- *Metabolic acidosis:* Critically ill patients due to conditions such as diabetic ketoacidosis, lactic acidosis, and renal failure may have associated metabolic acidosis. In these patients, as compensation, there will be increased carbon dioxide (CO_2) wash out to maintain acid–base balance. When these patients are intubated, even brief periods of apnea lead to a sharp increase in CO_2, which can dysregulate acid–base balance and worsen acidosis. Postintubation, the increased ventilator requirements may not be met due to lung-protective strategies as well as the limits of minute ventilation on the ventilator, which can lead to a drastic fall in arterial pH leading to cardiac arrest. Hence, these patients should be kept in spontaneous breathing as much as possible and intubation avoided, especially in whom ventilator cannot achieve the high minute ventilation requirement. A trial of NIV can be given to reduce the work of breathing during which correction of underlying acidosis can take place. If the patient needs intubation, then they should be maintained on spontaneous breathing both during intubation and on mechanical ventilation. Rapid sequence intubation is avoided, and if deemed necessary, a short-acting muscle relaxant like succinylcholine should be used. Postintubation, ventilator modes should be chosen that allow the patient to maintain their respiratory compensation. Modes like pressure control mode and pressure support mode, where the patient determines tidal volume and respiratory rate, is preferred. However, these patients with high-minute ventilation are at risk of developing relative hypoventilation, flow starvation, patient-ventilator asynchrony, and worsening acidosis.
- *Neurophysiologic derangement:* Patients with traumatic brain injury, stroke, or hemorrhage have an associated raised intracranial pressure (ICP). These patients may have low Glasgow Coma Scale (GCS), for which they need airway protection and, hence, may need intubation and mechanical ventilation. The process of laryngoscopy and intubation may lead to a further increase in ICP. Drugs used for induction can lead to a precipitous fall in blood pressure, which can be deleterious as it can lead to cerebral ischemia. Some of the measures that can be taken to prevent these complications are fluid loading to replete intravascular volume, especially in traumatic brain injury. Drugs like fentanyl and lignocaine can be used to mitigate stress response to laryngoscopy. It is prudent to avoid drugs like succinylcholine, which can increase ICP by causing fasciculations. Ketamine, which was thought to increase ICP, has not been proven in studies. Propofol and thiopentone sodium are associated with hypotension due to vasodilation and should be used with caution.
- *Hepatic derangement:* Patients with liver failure have associated hepatic encephalopathy and are at risk of raised ICP. They have associated coagulopathy, which makes them at risk for bleeding. Hence, intubation in these patients may be difficult, and measures to prevent raised ICP need to be taken.
- *Renal derangement:* Patients with renal failure may be associated with metabolic acidosis and hyperkalemia. Patients with metabolic acidosis will be tachypneic as part of the body's compensatory mechanism to maintain pH. Induction of anesthesia and subsequent positive pressure ventilation may lead to loss of compensatory response, leading to cardiac arrest due to further hyperkalemia. Drugs like succinylcholine should be avoided in these patients as it can increase serum potassium, leading to further hyperkalemia and cardiac arrest.
- *Gut dysfunction:* Patients in the ICU may have gastrointestinal problems like abdominal distension, paralytic ileus, ascites, pseudo-obstruction, obstruction, and raised intra-abdominal pressure. These patients are at risk of regurgitation and aspiration during laryngoscopy and intubation. Hence, these patients called as full stomach should be induced by rapid sequence induction. Fluid shifts in patients with paralytic ileus, pancreatitis, and intestinal obstruction may predispose them for hypotension during induction.

The physiologically difficult airway is more challenging than the anatomically difficult airway as the underlying conditions and factors are more life-threatening and difficult to manage. In an anatomically difficult airway, probably the safest and gold standard method of induction would be awake intubation;

however, in patients with physiologically difficult airway an awake intubation can worsen already altered physiology, like inadequate blunting of reflexes and sympathetic stimulation can worsen raised ICP, and cardiac ischemia. In patients with anatomically difficult airway, the primary aim is to ensure endotracheal intubation; however, in physiologically difficult airways the aim is to avoid complications and avoid further derangement in physiology. In an anatomically difficult airway, if endotracheal intubation and ventilation is not possible, then front-of-neck access is the ultimate option. However, in a physiologically difficult airway, there is no ultimate solution. One has to treat the underlying cause, as the time available is very limited. If not appropriately tackled, it usually ends up in a vortex, leading to cardiac arrest and death.

TABLE 3: Clinical examination of patients to look for anatomically and physiologically difficult airway.

S. No.	*Domain*	*What to examine*	*Comments*
1.	Head and neck	• *Facies:* Look for details that help to identify or suspect difficult airway, e.g., micro-/retrognathia, facial injuries, old scars, distorted anatomy, dressings, and secretions (nasal/oral) – *Cyanosis, severe pallor, and periorbital edema* – *Anxious, restless, agitation—delirium; difficult preoxygenation* – *Deviation of face, angle of mouth—stroke* • *Dilated neck veins*—increased intrathoracic pressure, chronic cardiorespiratory diseases (COPD and congestive cardiac failure) • Oral cavity—edentulous, oral hygiene, ulcers, bleeding, and candidiasis • Feeding tubes—a challenge for mask ventilation • Active ala nasi, accessory muscle use—poor lung mechanics, cardiorespiratory failure	General examination gives an idea of potential conditions or diseases that may increase the risk for difficult airway The examination can be focused—to identify the factors or conditions that may give clue for a physiological difficult airway (e.g., cyanosis—cardiorespiratory failure; periorbital edema—renal or cardiac failure)
2.	Extremities	• Clubbing, peripheral cyanosis—at risk of peripheral arterial disease • *Abnormal signs of tissue hypoperfusion:* Cold peripheries, prolonged capillary refill time • Peripheral edema—cardio, renal diseases, third spacing, and capillary leak (SIRS/sepsis) • Abnormal digits—deformities (rheumatoid arthritis—may have abnormal neck mobility) • Swollen legs—DVT/immobile/dependent state	Patients' extremities examination will add information of severity of physiological derangement or give a clue of diseases that may increase the risk of difficulty airway (e.g., neck mobility)
3.	Thorax and abdomen	• Asymmetry—kyphoscoliosis, parenchymal lung disease—patient may have reduced FRC and prone for cardiorespiratory adverse events • Tight dressings—trauma and postsurgery • Scars—midline—cardiac surgery, lateral—thoracic surgery: Potential risk of complications	Abnormal anatomy of thorax and paradoxical respiration are important findings to anticipate cardiorespiratory adverse events
4.	Bedspace	Monitoring devices, infusion pumps—vasopressor infusions, NIV machines, renal replacement therapy (RRT), and extracorporeal systems	Gives an idea on sickness of patients, organ dysfunction that help to choose drugs, and plan airway management
5.	Gastrointestinal system	• Recent recurrent vomiting and distended abdomen suggests high risk of aspiration • Conditions like gross ascites and ileus can suggest compromised respiratory function	Patients with abdominal pathology are at risk of aspiration

Contd…

Contd…

S. No.	***Domain***	***What to examine***	***Comments***
6.	Cardiorespiratory	• Shock and arrhythmias • Hypertension • Increased work of breathing and paradoxical breathing	Preexisting compromised cardiovascular state increases risk of peri-intubation complications
7.	Central nervous system	• Neurological deficits • Neuromuscular weakness • GCS and FOUR score	Intracranial pathologies influence the choice of drugs and technique to avoid complications

(COPD: chronic obstructive pulmonary disease; DVT: deep vein thrombosis; FOUR: Full Outline of UnResponsiveness; FRC: functional residual capacity; GCS: Glasgow Coma Scale; NIV: noninvasive ventilation; SIRS: systemic inflammatory response syndrome)

TABLE 4: MACOCHA scoring system for airway assessment.

Factors	***Points***
Patient factors: • Mallampati score 3 or 4 • Obstructive sleep apnea syndrome • Reduced mobility of the cervical spine • *Limited mouth opening:* <3 cm	 5 2 1 1
Pathology factors: • Coma • Severe hypoxemia	 1 1
Operator factors: Training—nonanesthesiologist	1
Score 0–12—0 = easy; 12 = very difficult	Total 12

TABLE 5: HEAVEN criteria for predicting difficult intubation.

Criteria	***Definitions***
H—Hypoxemia	Oxygen saturation value ≤93% at the time of laryngoscopy
E—Extremes of size	Pediatric patient ≤8 years of age or clinical obesity
A—Anatomic challenge	Trauma, mass, swelling, foreign body or other structural abnormality limiting laryngoscopic view
V—Vomit/Blood/ Fluid	Fluid present in the pharynx/hypopharynx at the time of laryngoscopy
E—Exsanguination	Suspected anemia that could potentially accelerate desaturation during RSI-associated apnea
N—Neck	Limited cervical range of motion due to immobilization or arthritis

(RSI: rapid sequence intubation)

Table 3 describes the clinical examination of patients, which gives clues to both anatomically and physiologically difficult airway.

Scoring Systems for Difficult Intubation in Intensive Care Unit

MACOCHA scoring: A scoring system is devised incorporating anatomical, physiologic, and operator characteristics and is validated for critically ill patients. This score allows for the recognition of patients with difficult intubation. **Table 4** describes components and scoring of MACOCHA score.

HEAVEN scoring system: A new scoring system called as HEAVEN scoring system has been recently studied, which incorporates both anatomical and physiological factors which can lead to difficult airway. This scoring system is being evaluated for use in emergency department; however for ICU, it needs to be validated. **Table 5** describes components and definitions of HEAVEN criteria for predicting difficult airway.

SUMMARY

Airway assessment in critically Ill patients is challenging. The tests available for difficult airway assessment lack sensitivity. Nevertheless, every attempt should be made to assess the airway when feasible, with complete preparedness to manage a failed airway. Critically ill patients have a physiologically difficult airway. The critical illness increases the risk of complications during tracheal intubation and transition to positive airway pressure ventilation. Physiological optimization of the patient and specific precautions need to be taken to minimize the risk of complications in these patients.

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Chapter

26

Examination of the Critically Ill with Point-of-Care Ultrasound

Kishore Pichamuthu

ABSTRACT

In this chapter, we discuss the wide range of point-of-care ultrasound (POCUS) applications and the different ways it can be performed while examining patients who are critically ill. We will also delve into some practical considerations while performing POCUS such as training, infection, control, and reporting.

INTRODUCTION

Point-of-care ultrasound (POCUS) has become an integral part of the evaluation and treatment of critically ill patients. Hence, it is important to know how to integrate it into the routine examination of such patients.

CASE SCENARIO

A 50-year-old lady was brought to the emergency department with a history of 3 days of fever and progressive breathlessness for a day and altered sensorium for 2 hours. In the resuscitation bay, she was found to be hypotensive and hypoxic. She developed bradycardia which progressed to an asystolic cardiac arrest. Advanced life support was initiated which resulted in a return of spontaneous circulation after 10 minutes. She was stabilized and shifted to the ICU. On arrival in the ICU, she is found to be intubated with a pulse oximetry saturation of 95% on 100% FiO_2 and 7 cmH_2O PEEP. She has crackles in both lung bases.

She is maintaining a mean arterial pressure of 65 mm Hg with 24 μg/min of noradrenaline infusion and 0.02 U/min of vasopressin infusion. The peripheries are warm. She is anuric and the blood lactate level is 4.6 mmol/L.

Her pupils are equal and reacting and she has a Glasgow Coma Scale of 2T/15 without the influence of any sedation.

It is evident that this is a complex critically ill patient and so a POCUS examination is performed.

We will return to this clinical case at the end of the chapter.

What is Point-of-Care Ultrasound?

Point-of-care ultrasound is a *focused* ultrasound examination performed at the *bedside* by *clinicians for diagnostic purposes* or *procedural guidance*.

It is focused as it tries to answer specific clinical questions and is not necessarily a comprehensive scan. It is a part of clinical examination (replacing and augmenting traditional methods)—not an investigation.

It may be performed at home, in transit, in outpatient areas, inwards, emergency rooms, ICUs, or operating rooms.

Any clinician who has had focused training and whose competence has been assessed by supervised scans can perform POCUS. The duration of training can vary from as short as 10 minutes to 10 hours of hands-on training, depending on the specific skill being taught.

The Rise of Point-of-Care Ultrasound

While descriptions of POCUS for cardiorespiratory assessments were initially published in the 90s, it is only in the last 15 years that this field has really taken off the ground. There have been many drivers for this change.

First, there has been an increase in research into new applications of POCUS in different domains. Many studies have established that POCUS findings change clinical management and that noncardiologists and nonradiologists can reliably screen for and identify abnormalities after a short focused training.

Second, ultrasound machines have evolved to become smaller and yet more capable. The rise of the ultraportables, which are truly pocket-sized machines with wired or wireless probes, has made the application of POCUS possible in more clinical areas than ever before.

Third, training in POCUS has become an integral part of most critical care, internal medicine, and emergency medicine training programs, leading to more practitioners being proficient in POCUS.

Finally, the coronavirus disease (COVID) pandemic pushed a wider application of POCUS, especially lung ultrasound because of the limitations of using traditional clinical examination [e.g., using a stethoscope while wearing full personal protective equipment (PPE)] and difficulties in using radiological investigations such as X-ray and computed tomography (CT) scans in patients with COVID acute respiratory distress syndrome (ARDS).

Point-of-care ultrasound is now an integral part of emergency and intensive care medicine. It has now become standard of care and it would be unethical to practice critical care medicine without it.

The Evidence for Point-of-Care Ultrasound

The use of POCUS has been shown to improve diagnostic accuracy. It has been demonstrated to have a high sensitivity for certain conditions like heart failure or pneumothorax compared with clinical and other radiological tests.

Studies have also revealed that POCUS helps confirm clinical diagnosis and leads to a change in diagnosis and/or management in a significant proportion of critically ill patients. It has also repeatedly shown to be cost and time efficient.

Evidence that POCUS improves hard patient outcomes like length of stay or mortality is lacking. This may be because of the varying methodologies for POCUS implementation and variations in the management decisions taken as a result of the POCUS examination.

Basics of Point-of-Care Ultrasound Examination

General Image Optimization

Irrespective of the area being scanned, a clear image with well-defined structures helps in making the diagnosis accurately. A wrong diagnosis because of poor imaging techniques will lead to wrong management decisions being taken and could have an adverse impact on patient outcomes. There are a few things that need to be done to optimize the image.

Patient Positioning

Optimal patient positioning makes it easy to acquire good quality images. For thoracic cardiac views, the patient should ideally be in a left lateral position. Subcostal imaging and abdomen and lung ultrasound require the patient to be in supine position. Supine or head-up up to 30° with a neutral head positioning is advisable for optic nerve sheath assessment and transcranial Doppler. Maxillary sinus ultrasound requires supine with head rotation to one side. Mild flexion at hip and knee joints with external rotation of the thigh aids in visualizing femoral and popliteal vessels. These are just some examples, and we need to be mindful of the position before we start scanning. In some situations, it may not be safe to reposition the patient, and the POCUS can be performed in the existing position itself.

Choosing the Right Probe and Mode

Most ultrasound machines available in the ICU have at least two probes. One high-frequency linear probe and another low-frequency phased array probe. Other probes like microconvex and large convex probes may also be used.

The frequency of the probes determines the resolution of the image and the depth of visualization. High-frequency probes offer high resolution, showing fine detail but image depth is limited to 5–6 cm. Hence, these are useful to scan superficial structures such as veins for vascular access, peripheral arteries, nerves, optic nerve sheath, and pleura. Low-frequency probes enable us to visualize deeper at the cost of fine detail. They are used for cardiac, abdomen, transcranial, and lung imaging.

Each probe may be used in several modes or presets. Choosing the right preset greatly improves image quality and avoids misdiagnoses. For example, the low-frequency phased array probe can be used for both cardiac and abdomen imaging. The cardiac preset offers a higher frame rate and thus better temporal resolution than the abdomen preset, which focuses on image smoothening with a lower framerate. Therefore, if cardiac imaging is performed with the abdomen preset, left ventricular (LV) contractility may appear sluggish due to the low framerate leading to a misdiagnosis of mild LV systolic dysfunction.

Depth and Gain Optimization

These two aspects should be optimized for all images to acquire clear images and to aid in correct interpretation.

Gain settings determine the amount of amplification of the raw ultrasound signal by the machine. Typically, fluid-containing areas appear anechoic, while tissues appear in various shades of grey. Optimal gain is when the contrast between the black of the anechoic areas and grey of the tissue reflections is maximum. To achieve this, the gain is increased till noise appears in the form of white specks in the areas that should be anechoic. It is then gradually reduced till the contrast between tissue and fluid is maximum **(Figs. 1 and 2)**.

The depth of the image is usually indicated on the right of the screen in centimeters. Depth should be adequate to ensure the visibility of the entire area of interest in the screen. Using more depth than required makes the area of interest appear smaller on the screen and in some machines leads to a lower frequency being used, compromising resolution.

View Specific Optimization

After all this general optimization, there are three steps in the process of acquiring good quality images prior to interpretation.

First is to make sure the probe position and orientation is correct.

Second is to verify that all the structures that are supposed to be visualized in that view are clearly seen.

Lastly, but very importantly, every view has some indicators of being a good view—the markers of quality. These need to be checked and if any of the quality marker criteria are not fulfilled, corrective adjustments need to be made to improve the image prior to interpretation. Given in **Table 1** are some positioning information and quality markers of commonly used views in torso ultrasound. While this includes echocardiographic views and abdominal and lung ultrasound, it is not a comprehensive guide and is offered here as an example of what needs to be looked at to optimize each view. It is out of the scope of this chapter to provide view optimization information for all ICU ultrasound applications.

Examination of Patients with Ultrasonography

Integrating Point-of-Care Ultrasound with Clinical Examination

Point-of-care ultrasound is not a separate investigation, but an integral part of the clinical examination. The two ways of practically integrating it are:

1. System-wise POCUS examination
2. Syndrome-based POCUS examination

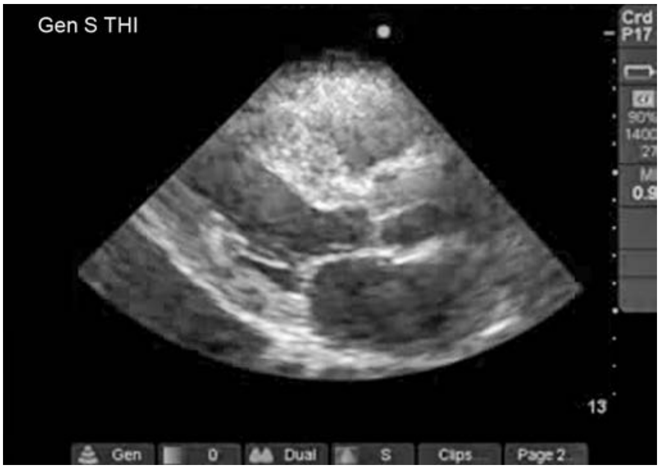

Fig. 1: An example of too much gain. Note the noise in the anechoic regions of the image.

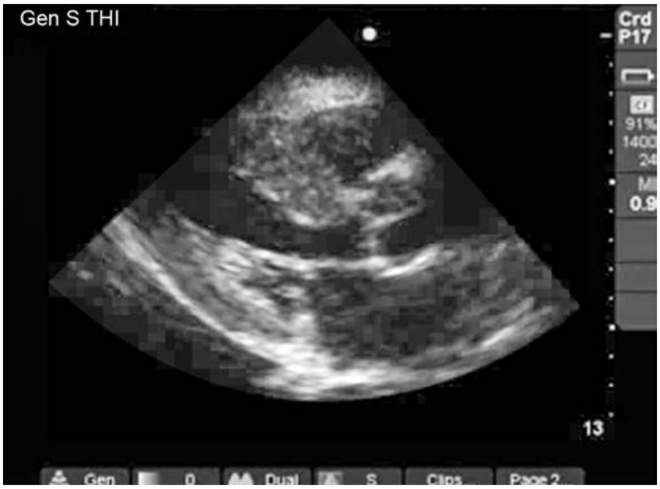

Fig. 2: Gain has been optimized from Figure 1.

System-wise pocus examination: One way to use POCUS as a part of clinical examination is to use it as a tool to examine each system side by side with other clinical methods. For example, if the respiratory system is being examined, then POCUS of the lungs is performed after auscultation. Or in cardiovascular examination, an echocardiogram is performed after assessing hemodynamics clinically.

Table 2 lists some of the abnormalities that can be looked for during POCUS of each system. This is not a comprehensive list but gives us an idea of what the scope of system-wise POCUs can be.

Syndrome-based pocus examination: In the last 20 years, several protocols have been described, which integrate cardiac, lung, and abdomen POCUS to try and elucidate

TABLE 1: Positioning information and quality markers of commonly used views in torso ultrasound.

Subcostal C4C

Probe position: 1 cm below xiphoid, midline, flat on abdomen, snugged up on the skin, and rocked toward left shoulder.
Dot marker position: Facing left
Structures: RA, RV, LA, LV, MV, TV, and AV

Quality marker	***Optimization maneuver***
Heart should be in the center of the image	Rocking movement of the probe
MV and TV should be seen opening in the same view. This is essential to accurately assessing relative sizes of LV and RV	Tilt the probe
No foreshortening	Rotate the probe
AV should preferably not be seen	Tilt probe downward—make the probe less flat
Visualization poor at >16 cm	Change to PEN (penetration)

PLAX

Probe position: Left sternal edge second to sixth space. Probe perpendicular to surface
Dot marker position: Facing right shoulder
Structures: LA, MV, LV, LVOT, AV, aorta, and RVOT

Quality marker	***Optimization maneuver***
LV horizontal (not tilted)	Stay close to sternum, as high intercostal space as possible
MV and AV opening in same view	Tilt the probe
No foreshortening	Rotate the probe
Darkening—due to rib	Slide the probe up or down

SAX

Probe position: Turn probe 90° clockwise from PLAX position
Dot marker position: Facing left shoulder
Structures: Three levels—*MV level:* LV, RV, and MV; *aortic level:* RA, TV, RV, PV, MPA, LA, and AV; *midpapillary level:* LV, RV, and papillary muscles

Quality marker	***Optimization maneuver***
True cross section, not an oblique view	Rotate the probe
Symmetry of the papillary muscles	Rotate the probe

A4C view

Probe position: Apex 5th ICS MCL
Probe directed: Dot marker faces left shoulder
Structures: RA, RV, LA, LV, TV, and MV

Quality marker	***Optimization maneuver***
LV apex in the center	Slide right or left movement of the probe
MV and TV opening in the same view	Rotate the probe. If the MV opening is not seen, then try flattening the probe on the skin (tilt). If AV is seen, reduce the flattening
No foreshortening	Slide down the intercostal space till LV is as long as possible

A5C view

Probe position: Apex 5th ICS MCL (same as A4C) but a slight clockwise rotation with a slight flattening of the probe toward the skin (tilt)
Probe directed: Dot marker faces left shoulder
Structure seen: A4C structure with AV and LVOT
Quality markers: Same as A4C

Contd…

Contd…

IVC

Probe position: Below xiphoid little to right of midline. Probe perpendicular to surface
Probe directed: Dot marker head ward
Structures: Liver window, tubular IVC, hepatic vein, heart, and aorta
How to confirm it is the IVC: (Important in severe hypovolemia to differentiate it from the aorta)

- Enter heart at right angles as opposed to aorta going tangentially behind
- Hepatic vein entry
- Visualize aorta separately

Quality marker	***Optimization maneuver***
Walls of IVC should be white and distinct	Tilt the probe
IVC should be as long as possible	Rotate the probe

LV systolic function using M-mode

View: PLAX. Ensure the papillary muscle is not in the view.
M-mode line just beyond the tips of the mitral leaflets. Junction of basal one-third and distal two-thirds.
Press M-mode button, acquire trace, freeze, calc, LV, measure LVD in diastole and systole (biggest and smallest)

Quality marker	***Optimization maneuver***
Septal and posterior wall endocardium should be distinct	
No posterior papillary muscle adjacent to the posterior wall	Tilt the probe
LV as horizontal as possible	Stay close to sternum, as high intercostal space as possible

Ascites

Probe position: Dependent parts of the abdomen (right and left lower quadrants and suprapubic). Perpendicular to the surface
Note: Minimal depth at the point of tapping >1.5 cm

Liver

Probe position:
Right lobe: Just below the costal margin MCL with beam directed posterior and superior
Left lobe: Midway between the xiphoid and the umbilicus
Dot marker position: To the left and tilt to sweep the right lobe and GB then place the dot marker to the head and tilt to sweep the right lobe.

Spleen

Probe position: Posterior axillary line 9–11th ICS
Dot marker position: Posterior and cranially. Tilt and rock probe to visualize the entire spleen

Kidney

Right kidney:

- Probe in right post axillary line two spaces below CP angle
- Liver is the window

Left kidney:

- Probe in left postaxillary line two spaces below CP angle
- Spleen is the window
- If gas obliterates it then go more posterior

Bladder

Probe position: Midline just above the pubic bone with the beam directed to the perineum
Dot marker: Head ward and to the left

Lung

Use echo/convex probe/microconvex in abdomen mode or high-frequency linear probe in superficial mode and switch off MB.
Probe position: Perpendicular to the ribs
Dot marker: Head ward

(AV: aortic valve; CP: costophrenic; GB: gallbladder; ICS: intercostal space; IVC: inferior vena cava; LA: left atrium; LV: left ventricle; LVD: left ventricular dysfunction; MB: multibeam enhancement; MCL: mid-clavicular line; MV: mitral valve; PV: pulmonary valve; RA: right atrium; RV: right ventricle; TV: tricuspid valve; LVOT: left ventricular outflow tract; RVOT: right ventricular outflow tract)

TABLE 2: System-wise POCUS.

Central nervous system	Optic nerve sheath ultrasound, transcranial sonography for midline shift, hydrocephalus, and transcranial Doppler
Head and neck	Maxillary sinusitis, cervical lymph node screening, carotid/jugular assessment, difficult airway risk and obstructive sleep apnea assessment
Lung	Pneumothorax, collapse, consolidation, pleural effusion, ARDS, and pulmonary edema
Cardiovascular	Left and right ventricular systolic function, regional wall motion abnormalities, left ventricular diastolic function, and LA pressures, RA/RV enlargement, pericardial effusion and tamponade, hypovolemia and fluid overload, preload responsiveness, and valvular abnormalities
Abdomen	Ascites, bladder distension, hepatosplenomegaly, solid organ abscesses, pyelonephritis and hydronephrosis, cholecystitis, and abdominal aortic aneurysm
Vascular	Deep venous thrombosis
Musculoskeletal	Septic arthritis, gout, fractures, myositis, muscle wasting, and diaphragmatic function

(ARDS: acute respiratory distress syndrome; LA: left atrium; POCUS: point-of-care ultrasound; RA: right atrium; RV: right ventricle)

the diagnosis in a problem-based manner. A few of these have been listed below:

- *Cardiac arrest:* FEER, CAUSE, and FEEL
- *Trauma:* eFAST and ABCDE
- *Respiratory failure:* BLUE protocol and RADIUS
- *Shock:* RUSH, SESAME, PIEPEAR, and SHOC-ED
- *Volume excess:* VExUS and FALLS

These are usually performed after clinical examination is completed and a syndromic diagnosis is made.

We will look at some of these in detail.

CAUSE: This stands for Cardiac Arrest Ultrasound Exam. This is done for patients with a no-shockable rhythm cardiac arrest. It consists of a subcostal 4-chamber view which is acquired during a 10 second pause in compressions. In this view, we screen for severe hypovolemia (empty ventricles), pulmonary embolism (RA/RV enlargement), massive myocardial infarction (noncontracting left ventricle), and cardiac tamponade. The lungs are imaged in the second intercostal space in the mid-clavicular line during breaths to rule out a tension pneumothorax.

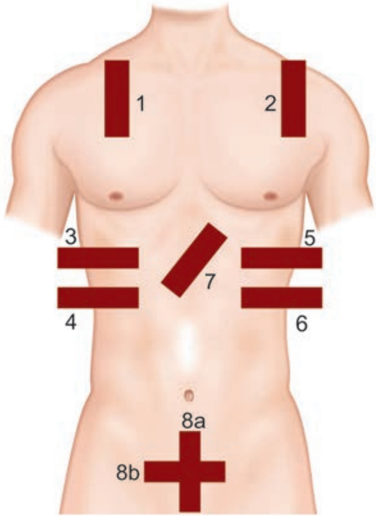

Fig. 3: The sequence of views that make up the eFAST (extended focused assessment with sonography in trauma) examination.

eFAST: This stands for extended Focused Assessment with Sonography in Trauma. It is used to detect pneumothorax, hemothorax, and hemoperitoneum in patients with penetrating or blunt chest or cardiac injury and penetrating or blunt abdominal injury. It can also be performed when the mechanism of blunt injury is not clear, or the trajectory of penetrating injuries is unknown.

The eFAST examination is performed immediately after the primary survey. It may have to be repeated within 10–15 minutes if the findings were not conclusive the first time.

The patient should be supine. Head-up tilt of the entire bed may improve visualization of fluid in the pleural spaces and the pelvis while a head down tilt will increase the sensitivity of finding fluid in the Morrison's pouch.

The convex abdominal probe or the echocardiography probe in abdomen mode can be used. These are low frequency probes (1–5 MHz). The same probe can be used for all the views.

Eight views need to be acquired and interpreted to complete the eFAST examination **(Fig. 3)**.

The first two views are in the third or fourth intercostal space in the mid-clavicular line bilaterally where we look for signs of pneumothorax (absent lung sliding, absent lung pulse, and absent B-lines). Then we move down to view the costophrenic angles for evidence of hemothorax. This is followed by the abdominal views to look for the presence of fluid in 4 Ps—perihepatic, perisplenic, pericardial, and pelvic.

Extended FAST is most useful in trauma patients who are hemodynamically unstable. A positive eFAST examination in such a patient necessitates an emergency laparotomy. In the hemodynamically unstable with a negative eFAST, it may be prudent to repeat it after 10 minutes. If a hemodynamically stable patient has a positive eFAST, a CT thorax and abdomen needs to be performed.

Extended FAST is limited, because it may not pick up retroperitoneal bleeds and penetrative solid organ injury. Clotted blood could be mistaken for soft tissue. Free fluid in the pleural, pericardial, and peritoneal spaces need not always be blood. Obesity and subcutaneous emphysema obliterate many of the views and reduce the diagnostic ability of eFAST.

In addition, POCUS can also be used to improve the primary and secondary surveys and this is summarized in **Tables 3 and 4**.

BLUE protocol: BLUE stands for Bedside Lung Ultrasound in Emergency. This is a POCUS protocol designed to assess patients with respiratory failure. Ultrasound is much more sensitive than clinical examination and chest X-ray for picking up lung pathology.

The low frequency cardiac or abdominal probe can be used. If available, a microconvex probe is ideal for lung ultrasound. If lung preset is available, it can be used, else the abdomen preset will suffice.

Three points on each hemithorax are insonated corresponding to the infraclavicular, mammary, and infra-axillary areas of auscultation. In each of these areas, we look for the presence of lung sliding, the presence of

TABLE 3: POCUS-assisted ABCD survey.

Airway	***Breathing***	***Circulation***	***Disability***
• Airway patency • Tracheal displacement/ compression • Prandial status	• Adequacy and equality of ventilation • Pneumothorax • Contusions • Pleural fluid • Diaphragmatic lesions • Multiple rib fractures	• *Echocardiography:* – Myocardial dysfunction – Pulmonary embolism – Cardiac tamponade – Valvular abnormalities • *Vascular ultrasound:* – IVC assessment – Aortic aneurysm – DVT • *Abdominal ultrasound:* – FAST – Retroperitoneal hematomas • *Soft tissue ultrasound:* – Pelvic, thigh hematomas – Peripheral vascular sufficiency	• *Cranial ultrasound:* – Raised ICP – Midline shift – Pupillary assessment

(DVT: deep vein thrombosis; FAST: focused assessment with sonography in trauma; ICP: intracranial pressure; POCUS: point-of-care ultrasound; IVC: inferior vena cava)

TABLE 4: Head-to-toes secondary assessment using POCUS.

Head	***Thorax***	***Abdomen***	***Extremities***
• Skull fracture • Maxillary fluid/hemosinus • Transcranial Doppler • Ocular injuries	• Lung contusions/lesions • Rib and sternum fractures	• Liver, spleen, kidney, and diaphragmatic lesions • Pneumoperitoneum • Retroperitoneal hematomas • Intestinal ischemia • Scrotal/testicular injuries • Fetal assessment	• Diaphyseal fractures • Muscular lesions • Soft tissue foreign bodies • Articular fluid • Soft tissue collections—hematomas/ pus

(POCUS: point-of-care ultrasound)

B-lines, and the presence of consolidation and pleural effusion.

Lung sliding is the motion artefact observed at the pleural line due to the movement of the visceral pleura over the parietal pleura. A-lines are the parallel horizontal lines seen below the pleural line in well-aerated lungs and pneumothorax. B-lines are rocket-shaped artefacts that arise from the pleural line and extend all the way down to the edge of the image **(Fig. 4)**. Consolidation appears like tissue (resembling liver) with air-filled hyperechoic bronchi within it **(Fig. 5)**. Pleural effusion appears as an anechoic space surrounding the lung, more so in the dependent areas **(Fig. 6)**. Pneumothorax is diagnosed when lung sliding is absent with absent lung pulse, absent B-lines, Barcode or Stratospheric sign on M-mode **(Fig. 7)** and presence of lung point **(Fig. 8)**.

Table 5 summarizes the diagnoses that can be made with BLUE protocol.

The BLUE protocol has been shown to be sensitive and can accurately identify the cause of respiratory failure correctly 90% of the time within 10 minutes.

RUSH protocol: RUSH stands for Rapid Ultrasound in SHock. It is a rapid assessment to determine the cause of circulatory failure. A low-frequency phased array probe is used for this examination.

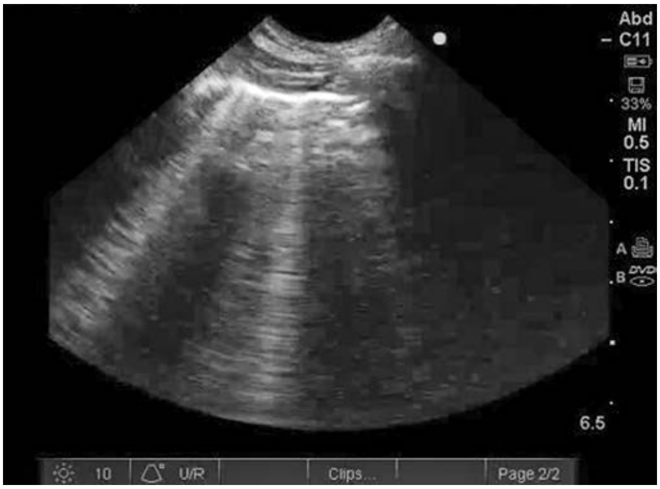

Fig. 4: Scattered B-lines can be seen.

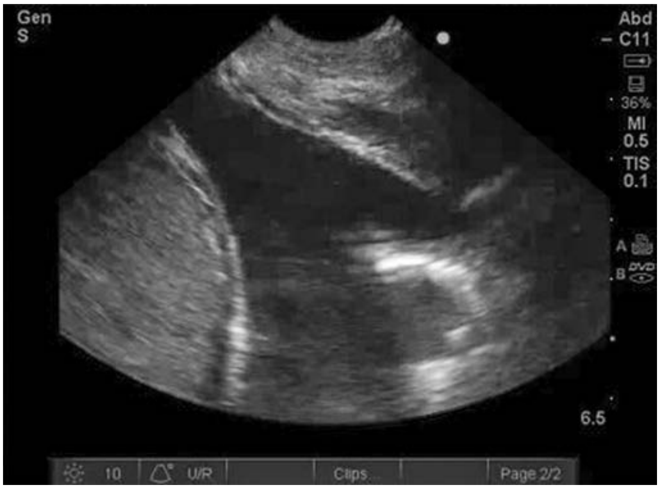

Fig. 6: Pleural effusion visualized in the costophrenic angle.

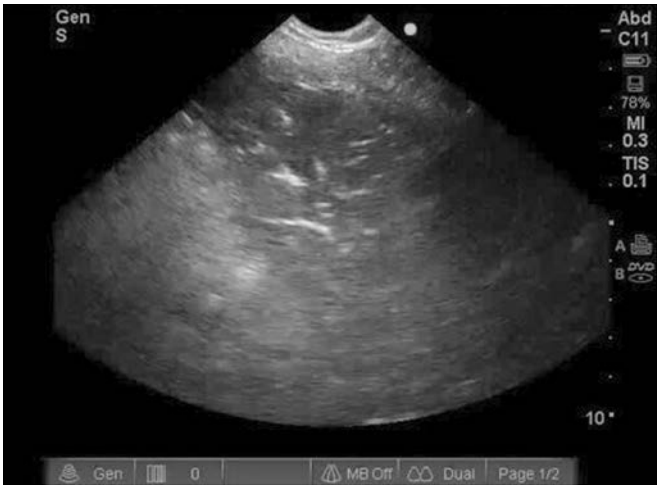

Fig. 5: Tissue-like appearance of lobar consolidation with hyperechoic air bronchograms.

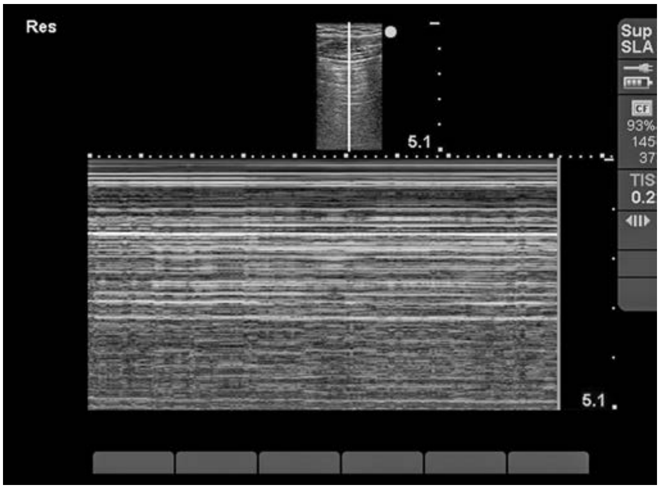

Fig. 7: M-mode examination in pneumothorax demonstrating the stratosphere or barcode sign, where the horizontal lines below the pleural line are as continuous as the lines above it.

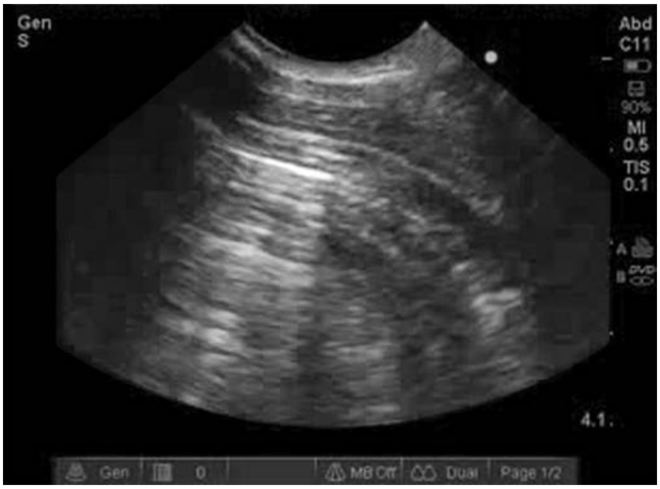

Fig. 8: Lung point where both signs of a pneumothorax and lung are seen in the same space.

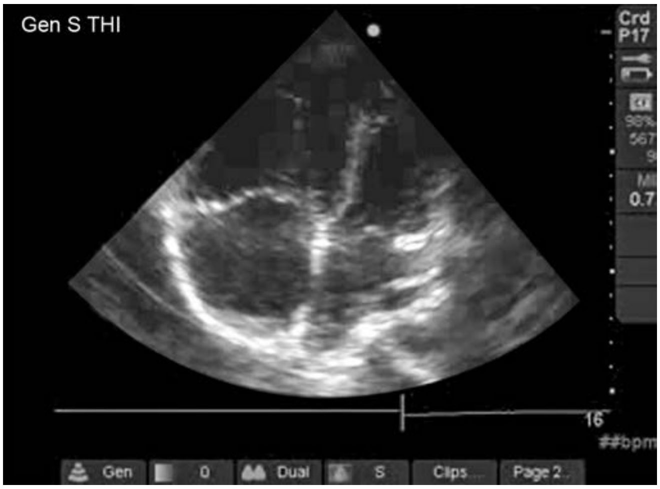

Fig. 9: Apical 4-chamber view showing massively enlarged right atrium (RA) and right ventricle (RV) in a patient with pulmonary embolism.

TABLE 5: Summary of BLUE protocol interpretation.

Lung sliding	***Pattern below pleural line***	***Diagnosis***
Lung sliding present	A-lines bilaterally, no DVT	Asthma, COPD, and pulmonary embolism
	A-lines bilaterally, DVT present	Pulmonary embolism
	B-lines bilaterally	Pulmonary edema, ARDS, and ILD
	Consolidation or focal B-lines	Pneumonia
Lung sliding absent	Lung pulse absent unilaterally, A-lines, lung point present	Pneumothorax
	Focal area of B-lines or consolidation	Collapse, pneumonia, and pleural adhesion
	Fluid in costophrenic angle	Pleural effusion

(ARDS: acute respiratory distress syndrome; BLUE: bedside lung ultrasound in emergency; COPD: chronic obstructive pulmonary disease; DVT: deep vein thrombosis; ILD: interstitial lung disease)

There are currently two schemata for the RUSH examination:

1. RUSH—HIMAP
2. RUSH—pump/tank/pipes

HIMAP is a mnemonic that refers to *H*eart assessment, *I*VC, *M*orrissons pouch/eFAST, *A*ortic assessment, and *P*ulmonary assessment.

We will look at the pump/tank/pipes approach as it is easier to remember in the heat of the moment.

The pump: The first step is cardiac assessment, which is referred to as the pump. Subcostal, parasternal long and short, and apical 4-chamber views of the heart are used to look for three specific things:

1. LV systolic dysfunction—qualitative and M-mode ejection fraction
2. RV dilatation with RV systolic dysfunction suggestive of an acute cor pulmonale **(Fig. 9)**
3. Pericardial effusion with tamponade—plethoric IVC with phasic RA and/or RV collapse

The tank: The second step is an assessment of the effective intravascular status, which is referred to as the tank. Three aspects of the tank are evaluated.

1. Fullness of the tank—heart chamber size, IVC size, respiratory variation, and internal jugular vein (IJV) collapse **(Figs. 10 and 11)**
2. Leakiness of the tank—eFAST to locate bleeding which is like a hole in the tank. Lung ultrasound to look for bilateral B-lines which might suggest fluid overload
3. Compromise of the tank—lung ultrasound to identify a pneumothorax

The pipes: The final step is an assessment of major arteries and veins termed as pipes, which may give us the etiology of hypotension. Two possibilities are screened for:

1. *Rupture of the pipes:* Screening for ruptured abdominal aortic aneurysm or dissecting aneurysm
2. *Obstruction of the pipes:* Screening for femoral and popliteal DVT with compression ultrasound

The RUSH protocol is a quick, easy to remember, and structured assessment of patients in shock.

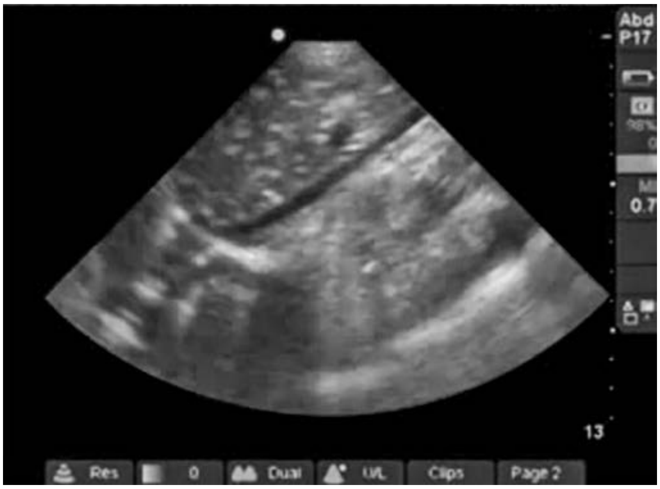

Fig. 10: A thin inferior vena cava (IVC) suggesting a low central venous pressure (CVP).

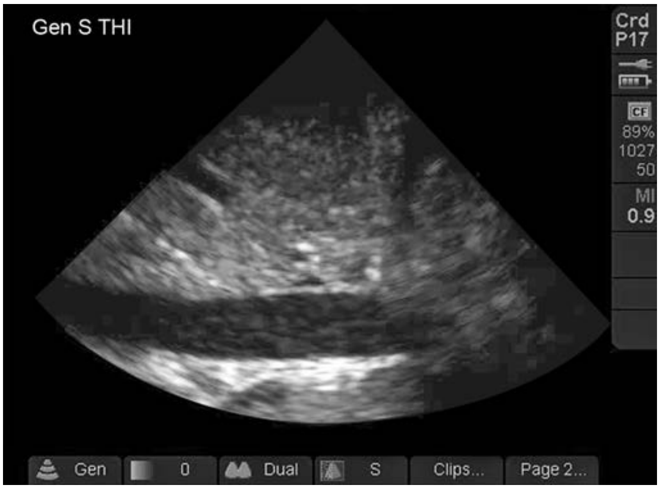

Fig. 11: A fat inferior vena cava (IVC) suggesting a high central venous pressure (CVP).

FALLS protocol: FALLS stands for Fluid Administration Limited by Lung Sonography.

It is a protocol that determines if it is safe to treat shock with fluid administration. It identifies patients with obstructive shock or cardiogenic shock with bilateral anterior B-lines or distributive shock with bilateral anterior B-lines as those in whom fluid resuscitation is unsafe.

VExUS: VExUS stands for Venous Excess Ultrasound. It combines IVC size with venous Doppler of portal, hepatic, and interlobular renal veins. The Doppler patterns are used to identify venous congestion and categorize it as mild or severe. It gives us useful information confirming a clinical diagnosis of fluid overload.

There are several other protocols describing the integration of POCUS into clinical examination. However, we have looked at some of the most popular. Describing all of them is out of the scope of this chapter.

Other Practical Considerations

Professionalism

- Whenever a POCUS examination is performed, the patient, if conscious, must be informed about the examination that is being attempted.
- Curtains should be drawn, and the privacy of the patient must be maintained.
- A chaperone is a must while examining patients.
- After the POCUS examination, the patient must be reassured or briefed about what the ultrasound showed. They are often anxious to know!
- The ultrasound gel must be wiped off the patient's skin, clothes must be replaced, and siderails pulled back up after the POCUS examination.

Ergonomics

The ultrasound examination should be performed from the side where it is comfortable for the clinician. This means that the sonographer stands on the side of the area to be examined. For example, while performing cardiac ultrasound, it is preferable to stand on the left side of the patient and do the scan with the left hand **(Fig. 12)** and while scanning a right pleural effusion, it is preferable to stand on the right of the patient and do the scan with the right hand.

Doing this allows the sonographer to stand straight without overstretching, reducing injury risk. It also reduces contact between the patient bed and the sonographer, which is good from an infection control point of view.

Infection Control

The ultrasound machine can become a vector contributing to the spread of multidrug-resistant organisms in the ICU. Prevention of contamination of the ultrasound machine and meticulously following a disinfection protocol will help prevent this.

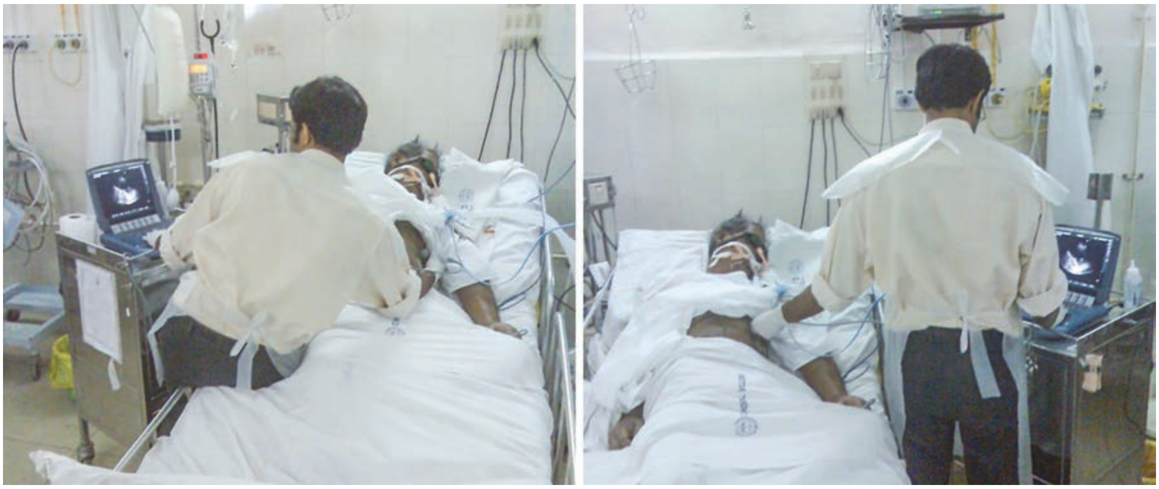

Fig. 12: Note the difference in posture and degree of contact with the patient and bed while performing cardiac ultrasound from either side of the bed.

Prevention/minimizing contamination of the ultrasound machine:

- The scanning hand should not be used to touch the ultrasound machine/keyboard
- The machine should not come in contact with the patients' bed.
- Use single patient package of ultrasound gel if available.
- After scanning, leave the probe on the patient bed till disinfected.

Disinfection Protocol

- The machine keyboard and probe holder should be cleaned with a manufacturer approved disinfectant.
- Remove gel from the probe.
- Disinfect the probe wire and then the probe before replacing it in the probe holder.
- Disinfect hands with hand-rub.

Documentation

It is very important to carefully document the results of the POCUS examination as it is the basis for management decisions and is useful for follow-up scans and comparisons.

Since it is a part of the clinical examination, the results can be documented with other examination findings and not as a separate investigation.

The date and time and indication must be mentioned.

Findings can be documented in a system-wise fashion or according to the POCUS protocol followed.

Important still images and video clips of the examination can be stored on the ultrasound machine, especially if they need to be shown to a senior clinician/specialist.

Training and Certification

It is important for all practitioners of POCUS to be adequately trained and certified. Training usually consists of hands-on training followed by supervised scans. The duration of training and the number of supervised scans required to achieve competency in POCUS depend on each individual skill that is being learnt. Training for POCUS has not yet been standardized. Currently professional bodies such as European Society of Intensive Care Medicine (ESICM) and the Indian Society of Critical Care medicine (ISCCM) are offering the European Diploma in Advanced Critical Care Echocardiography and the Fellowship in Critical Care Ultrasound, respectively.

Follow-up of Patient Case Scenario

Point-of-care ultrasound done according to the RUSH protocol revealed a plethoric inferior vena cava (IVC) and moderate LV systolic dysfunction. BLUE protocol lung ultrasound showed the presence of B-lines and subpleural microconsolidations in the dependent areas of both lungs. System-specific abdominal sonography picked up a bulky left kidney with dirty air shadowing. Transcranial Doppler revealed impaired diastolic cerebral flow in the middle cerebral artery.

In this scenario, POCUS has helped us to realize that this patient has a mixed septic and cardiogenic shock and has no obvious volume depletion. It has also helped us rapidly identify emphysematous pyelonephritis and cerebral hypoperfusion, both of which need immediate interventions. It also revealed a probable septic/ischemic myocardial dysfunction and delineated the extent of ARDS both of which influence the treatment plan. This is an example of how POCUS is invaluable in the evaluation of the patient in the ICU.

In this chapter, we have seen what POCUS is and how it can be incorporated into the examination of a critically ill patient. POCUS is an invaluable tool for this purpose, and it is imperative that we develop our expertise in this domain.

SUMMARY

Point-of-care ultrasound (POCUS) is a focused ultrasound examination performed at the bedside by clinicians for diagnostic purposes or procedural guidance. POCUS is now being extensively used in the day-to-day management of ICU patients. POCUS has been proven to improve diagnostic accuracy, influence management decisions, reduce costs, and increase patient safety. The prerequisites for good ultrasound image acquisition include correct choice of probe and preset, depth and gain optimization, and attention to view-specific quality markers in each image. POCUS can be integrated with clinical examination using a system-based approach or a syndrome-based approach. FEER, CAUSE, and FEEL protocols in cardiac arrest, eFAST and ABCDE protocols in trauma, BLUE, and RADIUS protocols in respiratory failure, RUSH, SESAME, protocols in shock and VExUS and FALLS protocols in fluid excess are examples of syndromic application of POCUS. We need to pay attention to professionalism, good ergonomics, infection control, and good documentation when performing POCUS in the ICU.

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Chapter

27

Examination of Patients on Extracorporeal Circuit

Rajesh Mohan Shetty, Rajavardhan Rangappa

ABSTRACT

Patients undergoing extracorporeal membrane oxygenation (ECMO) require a thorough, organized, and extended clinical assessment. It is often challenging to examine a patient on ECMO due to the complexity of the patient, equipment, monitoring devices, and limited space around the patient's bed. The clinical examination includes the ECMO device and its functionality and the patient's clinical examination. This chapter focuses on the structured way of examining a patient on ECMO, important findings to elicit, and meticulous examination of the ECMO device, ventilator, and bedspace.

INTRODUCTION

Clinical examination of patients on extracorporeal membrane oxygenation (ECMO) requires long, meticulous, and structured examination. It involves not only the clinical examination of the patient but also the ECMO device itself and its functions. It also includes careful monitoring for specific complications related to the ECMO.

CASE SCENARIO

A 45-year-old male patient was diagnosed with COVID pneumonia and severe acute respiratory distress syndrome. He is admitted in the intensive care unit (ICU) and was intubated in view of severe hypoxemia and respiratory distress. The patient continued to have refractory hypoxemia despite prone ventilation. The patient was initiated on venovenous (VV) ECMO with a drainage cannula in the femoral vein and a return cannula in the right internal jugular vein.

History and Symptomatology

After initiation on VV ECMO, the patient was able to meet oxygenation targets. On day 3, the patient was noted to have SpO_2 of 75% despite continuing ECMO support with rest lung ventilation.

How to Examine and Approach this Patient?

Examination

The examination of a patient on extracorporeal circuit can be done in a structured and sequential way:

- *Head-to-toe examination:*
  - Physical examination
  - Vitals
  - Sedation and analgesia
  - Cannulation sites
  - Laboratory parameters
- *Monitoring of ECMO device:*
  - Power supply
  - ECMO device
  - Flow settings
  - Circuit
  - Emergency kit
  - Pressure monitoring
  - Oxygenator
- *Monitoring for complications:*
  - Complications specific to VV ECMO
  - Complications specific to venoarterial (VA) ECMO

Head-to-toe examination

Physical examination: Routine system-wise head-to-toe physical examination of the patient is important to look for complications. Neurological examination of the patient with Glasgow Coma Score (GCS), pupils, and for any focal neurological deficit is crucial to rule out neurological complications. Patients should be ventilated at resting lung settings. Systematic reviews have found that ultralung-protective ventilation settings with tidal volumes of <4 mL/kg and a plateau pressure of <25 cmH_2O with positive end-expiratory pressure (PEEP) values between 5 and15 cm of H_2O are associated with a better outcome. Extracorporeal Life Support Organization (ELSO) recommends plateau inspiratory pressures below 25 cmH_2O, low $FiO_{2,}$ and PEEP between 5 and 15 cmH_2O. The patient should be monitored for any bleeding manifestations such as bleeding from the lung, gastrointestinal tract, genitourinary tract, and central nervous system.

Vitals: Monitoring of vitals of the patients on ECMO is similar to other ICU patients, but it differs in certain aspects like the oxygenation targets. ELSO recommends the saturation of 80% in VV ECMO patients. Monitoring of the vitals for the signs of hypoperfusion of the body in the form of cold limbs and/or reduced capillary refill in all the extremities and/or increasing lactates is important.

Monitoring for infection is very important in ECMO. However, it may be difficult to identify sepsis using the systemic inflammatory response syndrome (SIRS) criteria. Temperature is regulated by Hemotherm in ECMO patients; hence, fever may be missed. Most of the patients on ECMO may have features of SIRS responses because of interaction of blood with the ECMO circuit itself. However, it is very important to identify the sepsis by monitoring for subtle changes by holistic evaluation including changes in hemodynamics and elevation of inflammatory markers.

Advanced hemodynamic monitoring is fraught with challenge in patients on ECMO. Mixed venous oxygen saturation from the pulmonary artery gets oxygenated blood from the return cannula in VV ECMO giving erroneously high values. Indicator dilution method gives false results because of loss of indicator in the extracorporeal circuits. Monitoring of cerebral regional oxygen saturation (rSO_2) using near-infrared spectrometer (NIRS) might indicate global perfusion. Stroke volume variation will not be affected by VV ECMO and can be used to assess volume status. Ultrasonography/echocardiography can be used to assess the volume status and check RV function, lung aeration, and vessel patency.

Sedation and analgesia: It is important to assess the requirement for sedation and analgesia, especially in the early stages of ECMO. Opioids are considered the first line of medication for analgesia and sedation. They are also known to alleviate stress and decrease metabolic oxygen demand. Fentanyl is the most commonly used opioid. It has rapid onset and short duration of action but being a highly lipophilic drug, it is extensively consumed by the circuit and large doses are needed to maintain its effect. Various sedative agents such as benzodiazepines, propofol, and dexmedetomidine have been used. It is important to titrate these agents using reliable sedation assessment tool such as Richmond agitation and sedation scale (RASS).

Cannulation sites: Check cannulation sites daily to look for any bleeding or evidence of migration of the cannula. Also, examine the cannulated limbs to look for signs of deep venous thrombosis (DVT) and ischemia.

Laboratory parameters: It is recommended to routinely monitor the hemoglobin and platelet count. There are two schools of thought on targeting the transfusion threshold. Some experts recommend hemoglobin to be at normal levels or >10 g/dL, whereas evidence suggests that targeting hemoglobin of 7–8 g/dL might be enough to maintain adequate oxygenation. Patients on ECMO will be on anticoagulation to prevent the risk of clots in the circuit. Heparin is used commonly to target activated partial thromboplastin time (aPTT) ratio of 1.5–2 times.

Monitoring of ECMO device: It is important to do a complete structured examination of the ECMO machine during each shift. It includes the examination of the console, battery backup, gas supply, power supply, connection, and tubing.

Power supply: Make sure that the ECMO device is correctly plugged and the battery is fully charged. Every ECMO device will have indicators to show whether it is connected to the power source or is on battery backup. Make sure that it is connected to the power source and not on the battery backup.

ECMO device: ECMO flow depends on various factors such as preload, afterload, and dynamics of the circuit. Preload is determined by the patient's volume status, venous tone, size, length, number of holes, and location of the drainage cannula. Afterload is determined by the vascular resistance, position, size, and length of the return cannula. It is also important to know whether the machine is in free mode or intervention mode. In free mode when an alarm is activated, the ECMO machine will keep working whereas in intervention mode, it will stop. In intervention mode, it is important to have an ECMO specialist/perfusionist constantly at the bedside to act immediately.

Flow settings: Rotations per minute (RPM) determine ECMO flow on the machine. The higher RPM translates to higher flows. It is recommended to keep a flow of at least 2 L/min to avoid the risk of thrombosis.

Settings on the gas blender: The higher the FiO_2, the higher will be the oxygen delivery. It is kept at 100% initially and then titrated down according to the patient's requirements. Sweep gas flow will determine the CO_2 removal. The higher the sweep gas flow, the higher will be the CO_2 removal.

Circuit: It is important to check the cannula and circuit and make sure that no kinks are there in the entire length of the circuit. Make sure that there are ties at the defined places to make sure that cannulae do not migrate or get displaced. The entire length including the oxygenator and tubing should be inspected using a flashlight to check for clots or fibrins or breaks. There might be multiple stopcocks or tubing for monitoring pressures and sampling. Make sure that all of them are closed or connected to the appropriate places. Each circuit varies depending on the manufacturer; make sure that you are aware of the attachments/connections of the circuit for the machine you are using. It is also important to check the color of the circuit proximal and distal to the oxygenator to identify oxygenator malfunction and recirculation **(Fig. 1)**.

Emergency kit: Make sure that an emergency kit is available at the bedside. It should include clamps, emergency hand crank, additional circuits, and ties. Some large-volume institutes keep preprimed pumps as a backup.

Pressure monitoring: Monitoring pressures is one of the essential components of monitoring the ECMO. It is important to document pressures because more than the actual pressure, it is the trend in the pressure which will guide in identifying and managing any potential complications **(Fig. 2)**.

Venous pressure/P1: It is the pressure before the pump. Since the pump generates negative pressure to suck the blood, it is always negative. It should not exceed 100 mm Hg. A sudden rise in the negative pressure indicates flow insufficiency. It can be due to kinking or hypovolemia.

Arterial pressure/P3: It is the pressure after the oxygenator. It measures the pressures in the return cannulae. Generally, pressure should not exceed 200–250 mm Hg. A sudden rise in arterial pressure might indicate a kinked or an occluded return cannula. It may also happen due to patient-related factors such as movements or coughing.

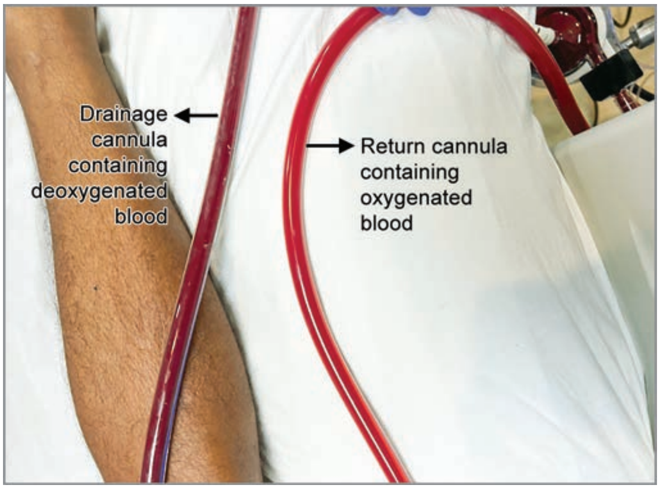
Drainage cannula containing deoxygenated blood
Return cannula containing oxygenated blood

Fig. 1: Tubing distal and proximal to the oxygenator showing difference in color corresponding to deoxygenated and oxygenated blood.

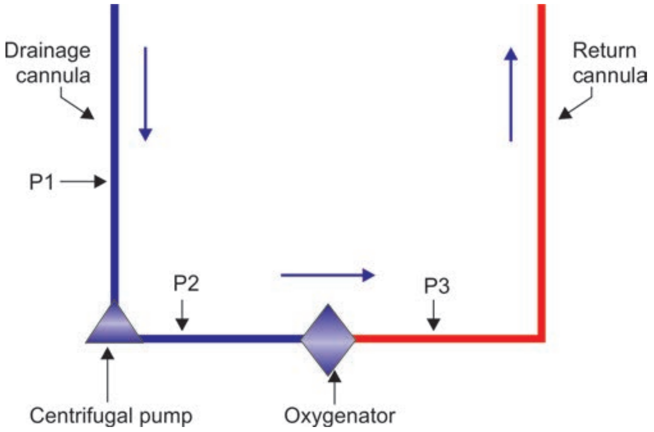

Fig. 2: P1—venous pressure, P2—preoxygenator pressure, and P3—arterial pressure representing pressures measured at different points in the circuit.

Delta P: It is the pressure difference across the oxygenator (between P2 and P3). It is usually <20 cm H_2O. It depends on multiple factors which include the type of oxygenator, coagulation parameters, and level of anticoagulation used. Delta P should not exceed >50 mm Hg; however, it is the trend of the pressure gradient that is important rather than a single value. It is measured by connecting pressure transducers before and after the oxygenator.

Oxygenator: The main functions of the oxygenator are oxygenation and CO_2 removal. Several different oxygenators are available in the market with different compositions, shapes, and shelf life. The function of the oxygenator can

TABLE 1: Complications specific to ECMO.

VV ECMO	*VA ECMO*	*Both VA and VV ECMO*
Recirculation	Ischemic limb	Hypoxemia
	LV distension in a patient with severely impaired LV dysfunction	Flow insufficiency
	Harlequin syndrome	Hemolysis
	Monitoring of native cardiac function	Oxygenator failure

(ECMO: extracorporeal membrane oxygenation; LV: left ventricular; VA: venoarterial; VV: venovenous)

be determined by visually inspecting for clots with the help of flashlight, measuring pressure across the oxygenator (delta P) and measuring the difference between pre- and postmembrane oxygenation. These clots can be classified into normal and abnormal clots. Normal clots are small fibrin clots at the top of oxygenators. Abnormal big clots can cause obstruction to the flow causing an increase in the transmembrane pressure gradient.

Monitoring for complications: It is important to identify and manage complications. These complications include flow insufficiency, hemolysis, blood clots, and oxygenator failure which are common to both VV ECMO and VA ECMO **(Table 1)**.

Flow insufficiency can be diagnosed by looking for chattering, high negative pressure, and fluctuations in the flow. It can be due to hypovolemia, drainage cannula migration, bleeding, and high flow.

Hemolysis can happen due to the spinning of red blood cells (RBCs) at high flow, oxygenator failure, pump with turbulent flows, and clotting in the cannulae. It is important to watch for this complication by looking at drop-in hemoglobin along with a dropping platelet count, change in color of the urine, and increase in plasma-free hemoglobin.

Complications specific to VV ECMO

Recirculation: Recirculation happens when a majority of the blood from the return cannula is sucked into the drainage cannula. The most common cause of recirculation is the close proximity of both cannulae with each other. Hypoxemia secondary to recirculation can be identified by looking at the color of circuit pre- and postmembrane along with monitoring oxygen in premembrane arterial blood gas which might be unusually high.

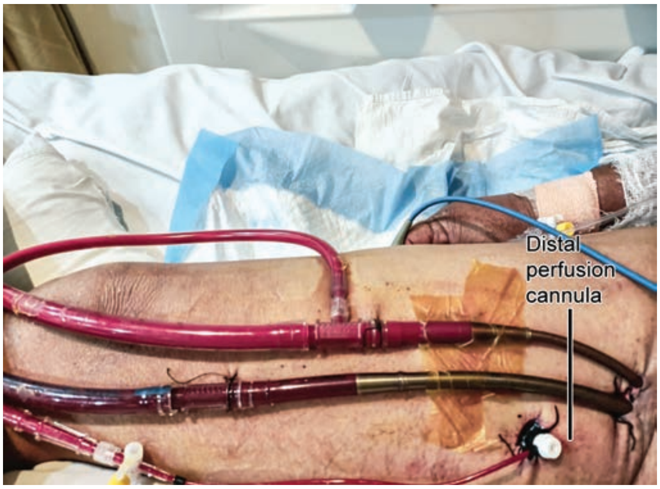

Fig. 3: Peripheral cannulation for venoarterial extracorporeal membrane oxygenation (VA ECMO) with a distal perfusion cannula.

Complications specific to VA ECMO

Ischemic limb: Features to look for include cold, clammy skin, increased capillary refill time, increased central to the peripheral temperature gradient, and mottling. This complication can be minimized by using a distal perfusion cannula and regularly monitoring the cannulated limb for oxygenation **(Fig. 3)**.

Left ventricular (LV) distension in a patient with severely impaired LV dysfunction: If LV afterload increases significantly, it will lead to LV distension, in turn leading to potential thromboembolic complications. Echocardiography should be used to measure not only LV function and chamber dimensions but also the extent and duration of aortic valve opening.

Harlequin syndrome: In VA ECMO, native cardiac blood flow mixes with retrograde blood flow from the femoral artery return cannula. In case of pulmonary dysfunction, poorly oxygenated native cardiac blood flow will perfuse the heart, brain, and upper limb, whereas richly oxygenated blood from the lower limb will perfuse the rest of the body causing differential oxygenation. It can be monitored by a pulse oximeter in the right-hand finger or by taking arterial blood gas samples from the right upper limb.

Monitoring of native cardiac function: Pulsatility in the case of VA ECMO indicates the presence of LV function and aortic valve opening. We should monitor for rhythm, pulsatility, and fluid responsiveness. The lesser the pulsatility or pulse pressure, the higher the chances of LV distension. Echocardiography assesses the native cardiac function using LV function and aortic valve opening. Advanced hemodynamic monitoring tools such as pulmonary artery catheter, PiCCO, FloTrac, and LiDCO are not validated for patients on ECMO.

SUMMARY

- Monitoring of patients on extracorporeal circuit involves monitoring of patient, monitoring of ECMO device, and assessment for complications.
- A structured approach is essential for routine examination of patients, identifying complications, and troubleshooting.
- Specific complications of VA ECMO are ishemic limb, Harlequin syndrome, and LV distension. Recirculation is a complication of VV ECMO.
- Cardiac output and other measurements using advanced hemodynamic monitoring devices such as PA catheters, PiCCO, FloTrac and LiDCO are not validated in patients on ECMO and may be inaccurate.
- Echocardiography still holds value for monitoring of patients on ECMO.

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Chapter

28

How will I Examine a Patient in a Prone Position?

Sai Saran, Mohan Gurjar

ABSTRACT

Prone ventilation is a life-saving intervention done in moderate-to-severe acute respiratory distress syndrome (ARDS). Like any procedure in the intensive care unit (ICU), it is associated with complications that can range from immediate life-threatening events such as accidental dislodgement of the airway to long-term sequelae in survivors such as loss of vision, neuropraxia, and pressure injuries. This chapter describes an organized approach to the examination of a critically ill patient in the prone position.

INTRODUCTION

Prone ventilation is a life-saving intervention done in moderate-to-severe acute respiratory distress syndrome (ARDS). In ARDS management, it is one of the strategies associated with a reduction in mortality along with low tidal volume mechanical ventilation. As is any procedure in the intensive care unit (ICU), it is associated with complications that can range from immediate life-threatening events such as accidental dislodgement of the airway to long-term sequelae in survivors such as loss of vision, neuropraxia, and pressure injuries.

This chapter describes an organized approach to the examination of a critically ill patient in a prone position.

CASE SCENARIO

A 36-year-old male was admitted to the ICU with a diagnosis of severe community-acquired pneumonia with circulatory shock. On day 2 of admission, his oxygenation worsened and required mechanical ventilation. Over the next 48 hours, his oxygenation did not improve despite ongoing clinical management and optimization of ventilator settings. The treating team decided on prone ventilation for ongoing severe ARDS.

Now, you have been asked to examine this patient who is undergoing third session of prone ventilation. How will you examine this patient?

History and Symptomatology Overview

It is necessary to know why the current prone positioning was done and to know the details of previous prone sessions, if done **(Table 1)**.

- *Current prone positioning:*
    - Indications/purposes
    - Types:
        - Spontaneous or positive pressure [noninvasive ventilation (NIV)/invasive mechanical ventilation (IMV)]
        - Awake or sedated/paralyzed
    - Duration of prone position planned
    - Manual or automated prone positioning
- *Previous prone positioning (if done):*
    - Total session
    - Median duration of prone positioning
    - Interval (in days) between ARDS onset and first prone session
    - Responder (early/late) or nonresponder
    - Persistent responder or not? Was he proned again within a few hours?
    - Any complications
    - Presence of any relative contraindication?

Clinical Examination of the Patient in the Prone Position

Examination during the prone position not only includes various aspects of the patient's physical examination but

TABLE 1: History and symptomatology overview of a patient required prone position.

S. No.	*To be reviewed*	*Reason*
1.	Indication for prone ventilation	To understand the pathophysiology, such as for hypoxemia or secretion clearance
2.	Interval (in days) between ARDS onset and first prone session	The earlier the proning, the better the response
3.	Pulmonary vs. extrapulmonary ARDS	As extrapulmonary better responds in the majority of cases
4.	• Type of respiratory support ongoing • Low dependency (nasal prongs/face mask)/medium (HFNO/NIV)/high (IMV)	Awake proning or under sedation-analgesia with paralysis: monitoring/duration aspects differ
5.	Any complications during the previous prone session	• Plans should be made for their prevention strategies or further aggravation of the injury such as facial swelling and pressure injury • If there is an arrhythmia, then better to target a high normal serum electrolytes range, etc.
6.	• Response to the previous session with session number and duration O_2 responder? • CO_2 responder? • Early/late responder • Sustained response to the prone session?	The duration of the current session can be planned based on that and further planning such as extracorporeal therapy initiation could be planned

(ARDS: acute respiratory distress syndrome; HFNO: high-flow nasal oxygen therapy; IMV: invasive mechanical ventilation; NIV: noninvasive ventilation)

also includes examination specific to the position itself **(Table 2)**.

General Physical Examination

- Patient's bed position and railings—horizontal, Trendelenburg position, reverse Trendelenburg position, and fall prevention measures.
- Head and neck position.
- Eyes protection (paddings and pupils).
- Airway and ventilator circuit—endotracheal/tracheostomy tubes and circuits, position, kinking, soiled, etc.
- Chest and abdomen—chest and abdomen are on a well-supported alternative pressure surface and ensuring bolsters placement if the abdomen is distended.
- Limbs position to avoid brachial plexus-related injuries
- Vascular indwelling catheter—presence/secured/strain, kinking [such as central venous catheter, arterial catheter, extracorporeal membrane oxygenation (ECMO)–related catheters, dialysis catheter, and temporary pacemaker]
- Nonvascular indwelling catheter—presence/secured/strain, kinking (such as feeding tubes, urinary catheter, core temperature probes, intercostal drainage, and surgical or percutaneous drains).
- General skin examination—review for pressure injury and ensure pressure points.
- Space and easy passage to the head end—ensure that there is adequate space at least 3 feet distance for easy access to the head end in case of any emergency
- Ensure that emergency equipment/drugs and manual resuscitator are available at bedside in case of any emergency.

Systemic Examination

Organ-specific assessment during current prone position:

Respiratory system:

- *Routine examination (inspection, palpation, percussion, and auscultation):*
    - Is patient with or without ventilatory support?
    - Presence of airway, mask, and NIV interface
    - Chest wall rise equally
    - Examine for drooling of saliva and oral hygiene
    - Palpation for the chest and neck for presence or absence of subcutaneous emphysema
    - Percussion could identify any hyper-resonant note (pneumothorax) or dull note (pleural effusions)
    - Auscultation can help in diagnosing endobronchial intubation, collapse/consolidation, airway obstruction, and pneumothorax

TABLE 2: Systematic examination in the prone position.

What to focus upon	***Rationale***
General examination	
The patient's bed position and railings	Easy access to the head-end in case of emergency
Head and neck position	Prevention of raised ICP, head end elevation of 10-20°
Eye protection	Prevention of ocular injury and complications such as loss of vision
Chest and abdomen	Review for the presence of thoracopelvic supports and any chance of raised IAP?
Position of limbs	Prevention of brachial plexus injury in upper limbs
Skin protection	Prevention of pressure injuries, the utility of alternative surfaces
Care of indwelling catheters	Prevention of accidental removal/dislodgment of both vascular and nonvascular indwelling catheters
Review of other paraphernalia required	Like equipment/manpower for handling the airway, to make the patient supine, in case of any emergency
Respiratory system	
Routine	• Look for airway/mask for any kinking, obstruction, soiling, position, etc. • Do routine respiratory system examination including inspection, palpation, percussion, and auscultation methods
USG chest	Follow PLUE protocol
Ventilator and respiratory mechanics	• Inspection of ventilator circuit, humidifier • Review ventilator settings • Assess respiratory mechanics
Arterial blood gases (ABGs)	Analyze ABG values and their changes due to prone position
Other organ specific examination	
Central nervous system	In clinical suspicion of raised ICP, look for ONSD of both eyes (after turning the head)
Cardiovascular system	• ECG lead placement and monitoring for ischemia and arrhythmia • Intravascular volume assessment including transhepatic IVC examination • Echocardiography assessment in prone position • Screen for DVT
Gastrointestinal system	• Feeding strategy to be planned • Monitor for worsening of intra-abdominal pressure
Renal system	• Look for decrease in urine output • In the presence of AKI, extravigilant for the indication for RRT

(ABG: arterial blood gas; AKI: acute kidney injury; DVT: deep vein thrombosis; GIT: gastrointestinal tract; IAP: intra-abdominal pressure; ICP: intracranial pressure; IVC: inferior vena cava; ONSD: optic nerve sheath diameter; PEEP: positive end expiratory pressure; PLUE: prone position lung ultrasonographic examination; RRT: renal replacement therapy; VILI: ventilator-induced lung injury)

- *Ultrasound specific examination:*
  - Areas for lung ultrasonography (USG) through "PLUE" protocol—prone position lung ultrasonographic examination **(Fig. 1)**:
    - Line 1: Paravertebral line (red color)
    - Line 2: Scapular line (green color)
    - Line 3: Posterior axillary line (yellow color)

    These are used to divide single side into three regions as shown in the **Figure 1**. Every region is divided into three equal parts to get nine areas out of which area over the scapular bone was excluded, leaving eight zones on each side totaling 16 zones to look for:
  - Look for "A" and "B" lines
  - Look for sliding sign, and rule out any pneumothorax which can happen as part of ventilator-induced lung injury (VILI)
  - Loss of aeration (collapse)
  - Positive end-expiratory pressure (PEEP) titration: B profile to A profile

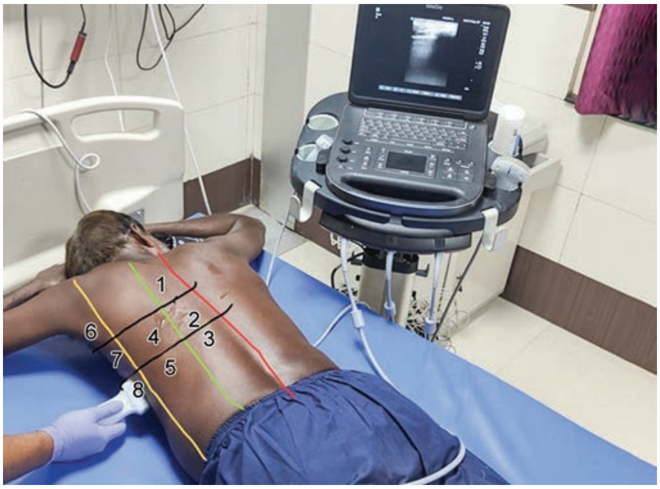

Fig. 1: PLUE—prone position lung ultrasonographic examination protocol: Zones of lung ultrasonography (USG).

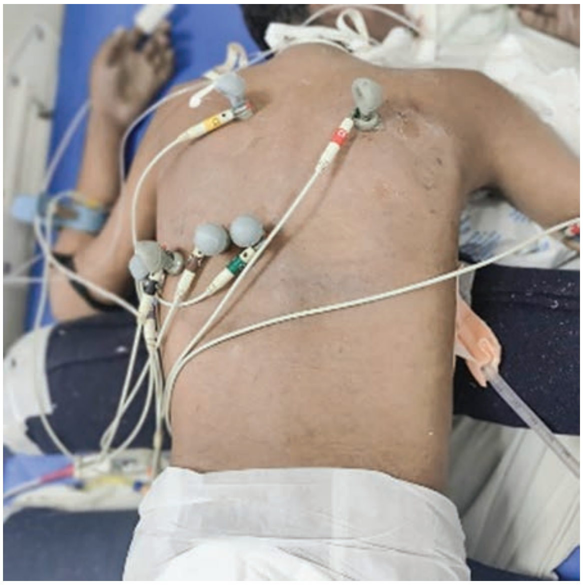

Fig. 2: Placement of electrocardiography (ECG) chest electrodes position and ECG of a patient in supine followed by prone position.

- *Ventilator circuit and machine specific examination:*
  - Invasive ventilation or NIV
  - Look for patient–ventilator asynchrony
  - Sedation adequacy (on invasive ventilation): Comment on sedation level such as Richmond agitation sedation score (RAAS).
  - Circuit: Any kink, leak, water, soiled etc.
  - Humidifier: Type, adequacy, and functional status
  - Ventilator supports (ongoing): Mode, pressure (peak, plateau, and PEEP pressures), tidal volume, respiratory rate, minute ventilation, driving pressure, compliance, FiO_2, etc.
  - Ventilator graphics and loops details
  - Assessment of lung mechanics: PEEP titration by assessing changes in compliance, plateau pressure, and driving pressure needs to be done to minimize VILI. Any suspicion of auto-PEEP can be diagnosed by pressing the expiratory hold button.
- *Arterial blood gas (ABG)-related examination:*
  - Review blood gases before and after prone positioning
  - Opine about benefit of prone positioning on the oxygenation and CO_2
  - PaO_2 responders are those whose PaO_2/FiO_2 ratio increases by at least 20% or by ≥20 mm Hg; whereas $PaCO_2$ responders are those $PaCO_2$ decreases by ≥1 mm Hg based on the available studies
  - In majority of the PaO_2 responders, the response will be early (within minutes to one hour) described as: "*Early responder*"; if this improvement in oxygenation takes up to 6 hours described as "*Slow responders*". Few patients might take many hours or more than one session to respond are described as "*Delayed responders*".
  - *Persistent responders* are those who show sustained improvement in oxygenation after the end of prone session; or a *nonpersistent responder* if otherwise. This can be reviewed from previous prone sessions.

Cardiovascular system:

- *Electrocardiography (ECG):*
  - Ensure that leads V_1 and V_2 were placed overlying the fourth intercostal space to the right and the left of the spine, respectively. Lead V_4 was placed in the fifth intercostal space on the left midscapular line. Leads V_3 and V_5 were positioned between V_2–V_4 and V_4–V_6, respectively **(Fig. 2)**. Lead V_6 and the limb leads remained unchanged from the standard position. There will be a decrease in mean QRS amplitude in leads V_1 to V_5 and reduction in R-wave amplitude in leads V_1 to V_4 on prone ECGs, with the precordial leads often demonstrating minuscule P waves and prominent Q waves in leads V_1 to V_3 due to changes in vectorial axes **(Fig. 3)**.
- *Invasive blood pressure:*
  - Examine and ensure proper leveling of the transducer to minimize the effect of hydrostatic pressure on invasive blood pressure measurements. For every 2.5 cm, the transducer is above or below the catheter level, the pressure in the system changes by 1.877 mm Hg.

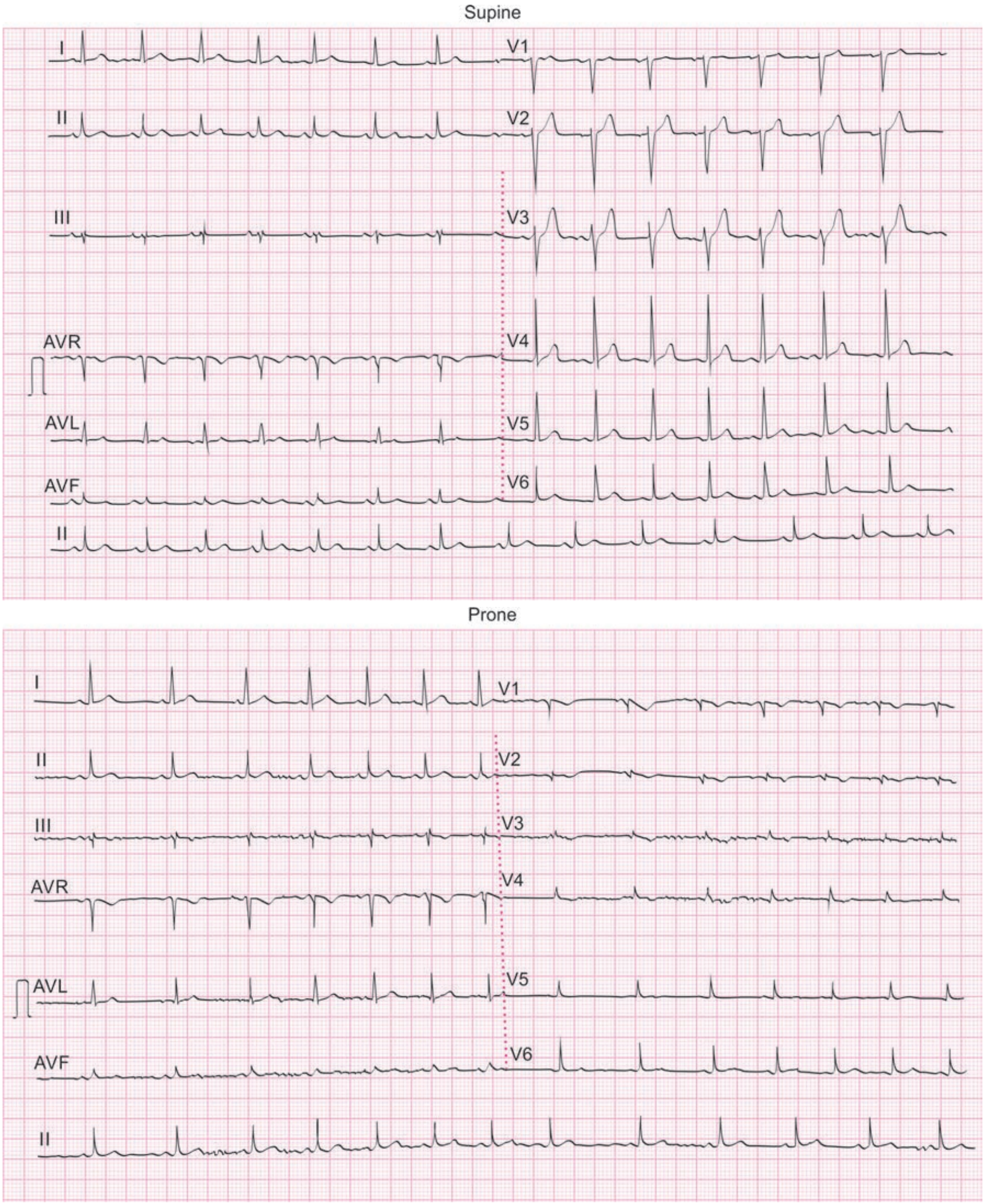
Supine
I
II
III
AVR
AVL
AVF
II
V1
V2
V3
V4
V5
V6
Prone
I
II
III
AVR
AVL
AVF
II
V1
V2
V3
V4
V5
V6

Fig. 3: Electrocardiography (ECG) changes from supine to prone position.

- USG assessment for intravascular volume/ echocardiography:
- Inferior vena cava (IVC) distensibility index could be calculated through subhepatic view; although defined cutoffs in this position are not available, trends of information might be useful. Fluid balance should be targeted to avoid the B profile in the lungs **(Fig. 4)**.
- *Echocardiography:*
  - Transthoracic echocardiography (TTE) could be done by temporarily deflating the lower thoracic section of the air mattress to position the probe

  optimally to obtain the apical four-chamber view. The operator is positioned to the left of the patient and uses his or her left hand to position the transducer. The only ultrasound window we could assess is the apical four-chamber view **(Fig. 5)**. The other way is to achieve swimmers' position, with the left shoulder slightly elevated by a pillow support. The transducer is placed along the left anterior axillary line at the fourth to fifth intercostal space with the indicator directed posteriorly to obtain the apical four-chamber view.
  - In cases of poor window, transesophageal echocardiography needs to be placed.
- *Cardiac output monitoring:*
  - Acute hemodynamic effects of prone position is dependent on many factors such as volume status (fluid responsive patients with increased cardiac output rather than fluid nonresponsive) and cardiac status pre-prone apart from response to prone session, with improved cardiac index in prone responders. Various gadgets based on thermodilution-based cardiac output such as PiCCO and LiDCO offer challenges due to alteration of phlebostatic axes.
  - Transducer-less systems such as transesophageal Doppler (TED) could be used to study the acute hemodynamic changes in cardiac output.

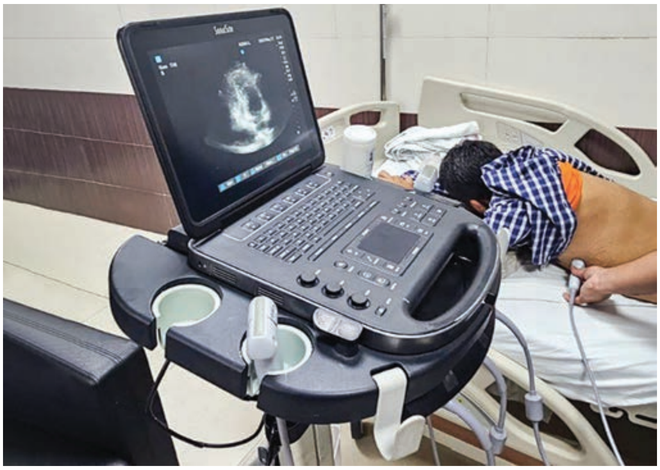

Fig. 4: Echocardiography in the prone position: Apical 4-chamber view using 2–8 MHz phased array probe.

Gastrointestinal system:
- Examine abdomen is free or not and review for the presence of thoracopelvic supports.
- Look for the presence of any regurgitated/vomited material.
- Look for ongoing tube feed (bolus or continuous) and feed acceptability during the prone session.

Renal system: Review urine output trends before and after prone positioning, as decrease in urine output might be simply because of kinking of urinary catheter or due to decrease in renal blood flow secondary to an increase in intra-abdominal pressure.

Other specific examination:
- *Optic nerve sheath diameter:* Assessment of raised intracranial pressure through measurement of optic nerve sheath diameter of one eye with head in lateral position and then other eye after turning the head, if clinically indicated **(Fig. 6)**.
- *Intraocular pressure (IOP):* As prone ventilation can lead to devastating complications such as loss of

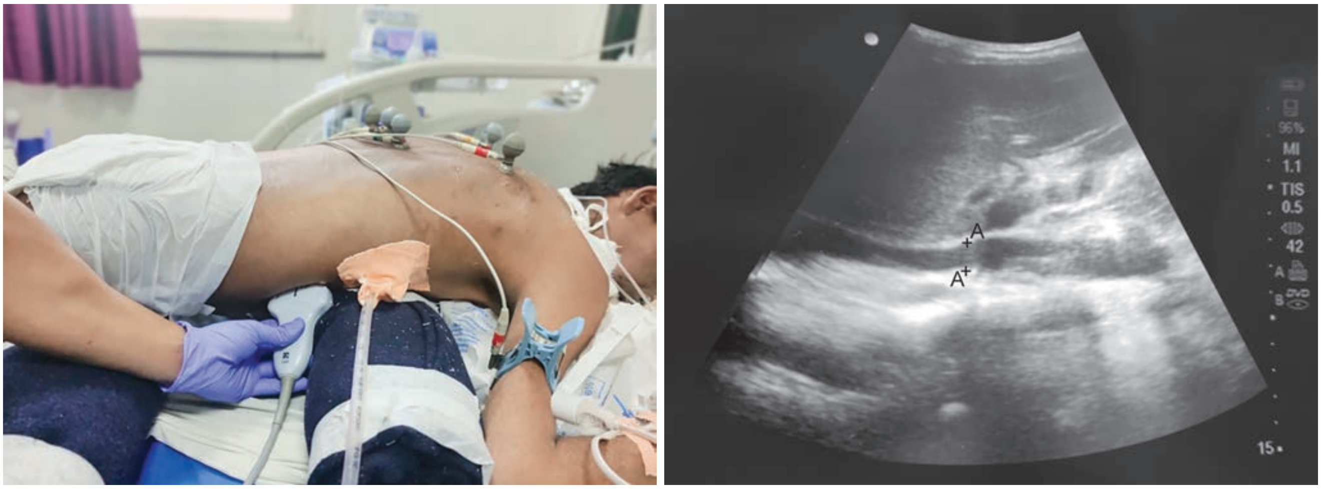

Fig. 5: Inferior vena cava (IVC) measurement in prone position using 2–5 MHz curvilinear probe.

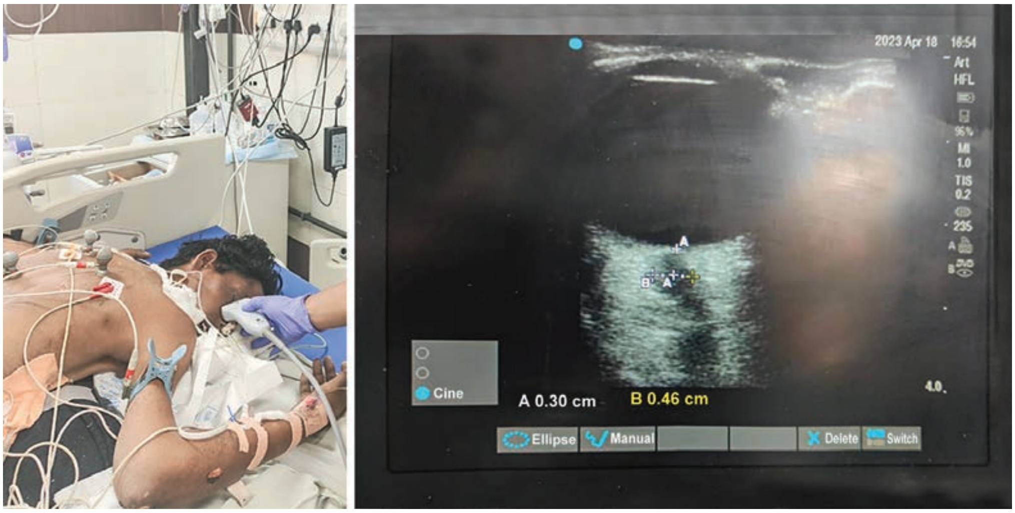

Fig. 6: Optic nerve sheath diameter (ONSD) measurement in prone position using 5–10 MHz linear probe.

vision, eye care is of utmost importance and IOP can be monitored especially in patients with glaucoma or other eye pathologies, which can lead to raised IOP.

- *Screening for deep venous thrombosis (DVT):* Deep vein screening, especially popliteal and axillary compression ultrasonography could be done easily in the prone position.

Specific Situations with Prone Position

- *Pregnancy:*
  - Pregnancy is a relative contraindication to prone positioning. The decision is usually being done after review with multidisciplinary team (MDT) involving obstetric, fetal medicine, infectious disease, pulmonary and critical care, and pediatric specialists.
  - Review and examine for appropriate padding (thoracopelvic supports) above and below the gravid uterus especially, if >24 weeks gestation to offload the uterus and avoid aortocaval compression, ensuring continuous cardiactocography (CTG).
  - This special condition requires trained and dedicated supporting staff.
- *Extracorporeal therapies:*
  - Few patients might require concurrent extracorporeal therapy such as continuous renal replacement therapy (CRRT) and venovenous ECMO. These should be initiated before prone positioning and can continue throughout prone session. Sometimes due to urgent indications for renal replacement therapy (RRT), there might be a requirement to place dialysis catheters in the prone position through USG guidance.
  - Assess and comment in brief about ongoing extracorporeal therapy, if not covered in any specific section/examination.
- *Cardiac arrest/chest compression:*
  - In case you encounter impending cardiac arrest during the examination, consider turning the patient to a supine position, and then early identification and corrective measures should be done at the earliest.
  - In any case if you have to start chest compression, remove thoracopelvic supports (if present), deflate the alternative pressure surface, and ensure head end is flat for effective chest compression. Studies reveal that the hand placement between T7 and T9 (central part of upper back at the inferior borders of scapulae) locates the compressions over the largest transverse section of the left ventricle for providing effective chest compressions.
  - Defibrillation (in case of ventricular fibrillation/ventricular tachycardia) and cardioversion for any unstable arrythmia are done by placement of pad in anterior-posterior (front and back) or pad/paddles in biaxillary (both armpits) position **(Fig. 7)**. Alternatively, defibrillator pads can be applied in the posterolateral position, that is, one pad in the left midaxillary line and a second one over the right scapula, or over the left scapula.

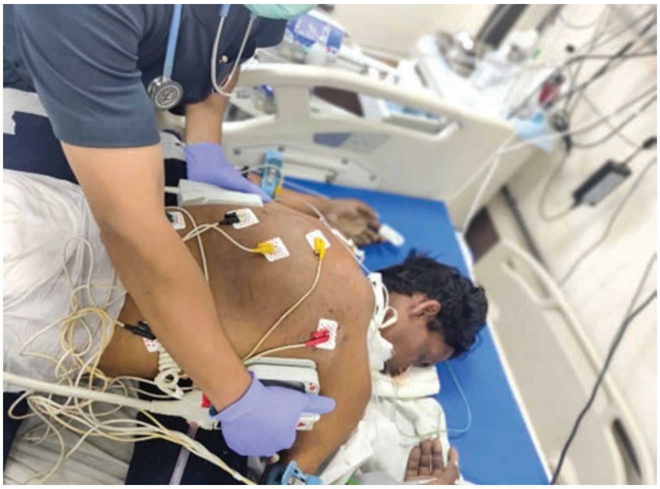

Fig. 7: Defibrillation in prone position by biaxillary pads placement.

SUMMARY

- Patient Mr/Mrs/age/gender
- Indication for prone position
- Duration of ongoing prone position session
- Your assessment for benefit in blood gases due to ongoing prone positioning
- Your assessment for respiratory system mechanics changes due to ongoing prone positioning
- Any issue/complication while making positioning for current prone session
- Any issue/complication after positioning for the current prone session.

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Chapter

29

How will I Examine a Patient with Unknown Poisoning?

Binila Chacko, Lovely Thomas

ABSTRACT

This article is an introduction to the fundamental concepts underlying the initial approach to the patient who has been poisoned. Regardless of the perceived toxidrome that may be encountered, supportive care should be administered immediately to any patient who displays symptoms of toxicity. The focus of this chapter is to assist trainees to do a quick history and clinical assessment to facilitate listing out possible agents responsible for the clinical presentation. This in turn will help in ensuring appropriate therapy and antidotes if indicated.

INTRODUCTION

Critical care physicians frequently treat patients who have been poisoned. Patients may unintentionally or purposefully be exposed to dangerous poisons. Dose, chemical characteristics, how quickly the victim presents themselves for medical attention, and the victim's baseline health all have a role in the result of poisoning. Most cases of poisoning can be saved with prompt diagnosis and the start of appropriate supportive therapy. This article discusses the initial approach that a clinician must take to help narrow down a diagnosis.

CASE SCENARIO

At about 12 noon, friends rushed an 18-year-old student to the emergency department (ED) after finding her in her hostel room disoriented, drowsy, and incontinent. She was last seen well 12 hours prior. She had a history of psychiatric illness and has been treated with fluoxetine, propranolol, lithium, clozapine, and lorazepam. She had a history of systemic lupus erythematosus for which she was on immunosuppression.

On examination in the ED, she was febrile with a temperature of 102°F, heart rate was 62 beats/min, systolic blood pressure (BP) 110/70 mm Hg, and respiratory rate 22 breaths/min. She was confused and delirious and her Glasgow Coma Scale (GCS) was 13/15. There was no evidence of neck stiffness or focal neurological deficits. Pupils were normal size with equal reaction to light. She had hyperreflexia in both upper and lower limbs and there was ankle clonus. Rest of the systemic examination was normal. How will you approach this case?

Initial Approach to the Patient

Initial examination in any poisoned patient should be prioritized to identify life-threatening physiological or clinical abnormalities. As in any critically ill patient, assessment and management should go in parallel with focus on the components of ABCDE (airway, breathing and circulation, disability, and exposure).

Airway: Several factors may compromise the airway in patients with poisoning and broadly can be due to the inability to protect the airway or airway obstruction **(Table 1)**. A systematic approach would help determine the cause of airway obstruction. Airway compromise should be suspected in any patient with a decreased level of consciousness and noisy respiration. Priority would be given to securing the airway if measures such as clearing oral secretions, insertion of an oral airway, and the triple maneuver fail to restore airway patency.

Breathing: The adequacy of breathing may be compromised in poisoning due to several factors, which must be meticulously determined. Poisoning can result in both type I (due to V/Q mismatch) and type II respiratory

TABLE 1: Causes of the compromised airway in a poisoned patient.

Inability to protect the airway due to reduced consciousness	***Airway obstruction***	
	Intrinsic causes	***Extrinsic causes***
Direct CNS depression (e.g., OP, sedatives, hypnotics, alcohol)	Excessive oral secretions (e.g., OP, pyrethroid compounds), vomiting, oral bleeding	Direct (hanging, trauma)
Seizures (e.g., pyrethroids, organochlorine compounds)	Tongue falling back (due to low GCS)	Indirect (subcutaneous emphysema)
Cerebral edema (secondary to hypoxia, hypotension)	Laryngeal edema (hair dye poisoning, postintubation)	
Head injury (secondary to fall)	Laryngeal dysfunction (e.g., OP)	

(CNS: central nervous system; GCS: Glasgow Coma Scale; OP: organophosphorus)

failure (hypoventilation due to neurodepressive effects of the poison combined with hypoxia, hypotension, metabolic effects, and fatigue due to increased work of breathing). Assessing the respiratory rate, use of accessory muscles, and respiratory effort should be the primary focus of the clinical examination, which should also include measuring oxygen saturation and estimating arterial blood gases (ABGs). A plain chest radiograph would aid in diagnosing aspiration. Noninvasive ventilation (if the patient can protect the airway) or invasive ventilation may be used to support breathing.

Circulation: Depending on the toxicity patient is exposed to, they may manifest with hypotension, hypertension, tachycardia, or bradycardia. Overdosing on an organophosphorus (OP) compound, sedatives/hypnotics, serotonergic drugs, or cholinergic substances often leads to hemodynamic consequences. Clinical examination should focus on the assessment of perfusion (skin, urinary and neurologic windows, although neurological dysfunction may be due to direct effects of the toxin on the brain), basic and advanced hemodynamic assessment (when indicated), and investigations [electrocardiogram (ECG), cardiac enzymes, echocardiogram (ECHO)].

Disability: Either the GCS or the AVPU (alert, vocal responsive, pain responsive, unresponsive) scale should be used to evaluate the neurological status of the patient. Significant loss in consciousness indicates strong neurodepressive effects of the toxin, with or without serious organ malfunction (respiratory, hemodynamic, or metabolic). Pupillary inspection may provide diagnostic clues **(Table 2)**. Focal neurological symptoms should prompt the doctor to consider an alternate diagnosis despite the fact that focal symptoms may occasionally be observed in overdose.

Exposure: The patient's clothing should be removed (bearing the dignity of the patient in mind) and a thorough examination should be performed looking for signs of trauma, bleeding, and needle marks suggestive of drug abuse.

Once the patient's condition has been stabilized, the attending physician's attention should turn to narrowing the list of differential diagnosis and confirming the diagnosis. The identification of symptoms, signs, laboratory findings, and ECG changes that define a particular toxicologic syndrome, also known as a toxidrome, can help narrow a differential diagnosis to a particular class of poisons and direct subsequent management **(Table 2)**. However, it is important to rule out other important differentials such as an acute central nervous infection, seizures, or sepsis from another focus.

History and Symptomatology

It is important to get a good history of the current illness. While information can be gained from the patient if he or she is aware and conscious, in most cases of purposeful ingestion, the patient's mental state renders the history untrustworthy, and there may be further difficulties in the context of a patient with a depressed sensorium. In such cases, history can be taken from collateral information obtained from family, friends, or medical records looking for a history of psychiatric illness. The history must include the time, route of entry, quantity, intentional or accidental exposure, availability of drugs at home, and whether any member of the family has chronic diseases (such as

TABLE 2: How to make a quick diagnosis of the toxidrome through clinical examination?

Toxidrome	***Onset***	***Temperature***	***Sweating/ secretions***	***Sensorium***	***Pupils***	***Tone***	***Reflexes***	***Rhabdomyolysis***	***HR and BP***	***Bowel sounds***
NMS	Delayed 1–3 days	Hyperthermia	Increased	Agitated	Normal	Rigid	Normal	+++	Labile	–
Sympathomimetic	Rapid	Hyperthermia	Increased	Agitated	Mydriasis	Increased	Exaggerated	++	Increased	Increased
Serotonin	Rapid	Hyperthermia	Increased	• Agitated • Confusion in the late stage	Mydriasis	Rigid (severe toxicity)	• Exaggerated • Ankle clonus and opsoclonus (mild-to-moderate toxicity)	++	Increased/ labile	Increased
Anticholinergic	Rapid	Hyperthermia	Decreased	Agitated	Mydriasis	Normal to increased	Normal to increased	–	Increased	Decreased
Malignant hyperthermia		Hyperthermia	–	Depressed	Normal	Rigid	Depressed	++	Increased	–
Cholinergic	Rapid	Normal	Increased	Agitated Depressed	Miosis	Decreased to normal	Depressed to normal	–	• Decreased • Increased in a minority	Increased
Opioids	Rapid	Hypothermia	–	Depressed	Miosis	Normal	Normal	–	Depressed to normal	Decreased
Sedatives	Rapid	Hypothermia	–	Depressed	Normal	Normal	Normal	–	Depressed to normal	–

(BP: blood pressure; HR: heart rate; NMS: neuroleptic malignant syndrome; + to +++ indicates the increasing impact of effect; – indicates no known effect) hypertension, diabetes, etc.) and whether missing tablets or any empty pill bottles or other material were found around the patient.

A careful and systematic approach to clinical examination can help narrow down the toxidrome. In this article, the focus will be on making a quick identification of common toxidromes—cholinergic, sympathomimetic, serotonin syndrome, opioid overdose, sedative overdose, neuroleptic malignant syndrome, and malignant hyperthermia—with this approach.

General Physical Examination

A quick head-to-toe examination must be done in every poisoned patient. Given below is a nonexhaustive list of possible agents linked to the general examination findings.

- *Breath odor:*
  - Fruity—isopropanol
  - Garlic—OP compound and arsenic
  - Glue—toluene
  - Fishy—zinc phosphide
- *Perioral ulcerations, mucosal erythema:*
  - Corrosive agents—acid and alkali ingestion
  - Paraquat tongue—erythematous swollen tongue later followed by erosions and ulceration
- *Face and neck:*
  - Ligature marks—for attempted hanging
  - Angioneurotic edema—Super Vasmol poisoning
- *Skin color:*
  - Erythema and edema—carbon monoxide poisoning (cherry red color seen with 2% of severe carbon monoxide poisoning), boric acid
  - Cyanosis—methemoglobinemia, cyanide poisoning, aniline dyes, nitrites, nitrates
  - Blisters—barbiturate poisoning
  - Dry—anticholinergic syndrome and antihistamine overdose
  - Diaphoretic—cholinergic syndrome, neuromalignant syndrome, serotonin syndrome, and sympathomimetic syndrome
  - Hyperkeratosis and melanosis—chronic poisoning of arsenic
- *Nails:* Mees lines—arsenic, thallium, and fluorosis
- *Injection marks/tattoo:* Suspect intravenous drug abuse
- *Nasogastric aspirates:*
  - Altered blood—iron ingestion and corrosive ingestion
  - Blue—copper sulfate
- *Urine examination:*
  - Calcium oxalate crystals—ethylene glycol poisoning
  - *Color:*
    - Orange to red-orange color—rifampicin, deferoxamine, mercury, or chronic lead poisoning
    - A brown color with chloroquine or carbon tetrachloride
    - Greenish blue color with copper sulfate, phenol poisoning, or methylene blue toxicity
  - Blood positive with no red blood cell (RBC) in urine—suspect methemoglobinemia
- *Color of blood:* Chocolate brown—methemoglobinemia

Assessment of the core temperature is important in every poisoned patient.

The core temperature must be at least 38.3°C for a diagnosis of drug-induced hyperthermia. *In the case scenario, the patient was hyperthermic.* It is important to eliminate organic sources of fever, such as infectious causes and noninfectious causes such as heat stroke, thyrotoxicosis, and phaeochromocytoma. Peripheral factors contributing to hyperthermia include:

- *Increased heat generation, either due to:*
  - Increased heat production due to muscular hyperactivity (as seen with neuroleptic malignant syndrome, anticholinergic, sympathomimetic, and serotonin poisoning and malignant hyperthermia) or seizures or
  - Increased metabolic rate (as seen with salicylate overdose)
- Slower heat loss due to impaired heat dissipation with anticholinergic drugs

Differentiating between these syndromes is aided by the history of offending medication as well as the chronological relationship to ingestion and other associated clinical features. *Time to onset* of hyperthermia can help differentiate neuroleptic malignant syndrome (NMS) from the other syndromes. In NMS, time to onset of symptoms is gradual over 1 to 3 days as opposed to the other syndromes when the toxidrome onset is rapid.

In general, hypothermia in a poisoned patient is associated with opioids, sedative-hypnotics, anesthetic medications, and sympatholytic (beta blockers and clonidine) overdose.

Systemic Examination

Neurological Examination

As mentioned earlier, it is paramount to rule out non-toxicological causes of this patient's altered behavior. If there is a suspicion of meningitis or encephalitis, a lumbar puncture must be done once raised intracranial pressure is ruled out (fundus examination and imaging), even if there is no clinical evidence of the same given the reports of limited diagnostic accuracy for clinical characteristics (reported sensitivity of Kernig sign 57% and Brudzinski sign 97%). If there is a suspicion of a cerebrovascular accident, it is preferable to do a magnetic resonance imaging (MRI) brain over a computed tomography (CT) scan to rule out a brainstem stroke.

In general, a depressed sensorium can be seen in patients with overdose of opioids, sedative-hypnotics, alcohol, antipsychotics, cholinergic, sympatholytic, antiepileptics, and hypoglycemic agents. In contrast, sympathomimetics, anticholinergics, serotonin syndrome, salicylates, and central hallucinogens can result in an agitated state.

Examination of the pupils can further help in the following manner. Normal pupillary size ranges from 2 to 4 mm in bright light. A person is said to have miosis if the pupillary size is <2 mm and mydriasis if >4 mm.

Sympathomimetic, anticholinergic, and serotonin syndrome physiologically will have mydriasis, whereas opioids, cholinergic, and sympatholytic overdose will have miosis. Meperidine overdose is an opioid outlier and is not associated with miosis. Additionally, this overdose typically causes seizures over a depressed sensorium.

Assessment of tone and deep tendon reflexes is also important in an unknown poisoning. The presence of limb rigidity could alert the physician to the common possibilities of NMS, serotonin syndrome (severe toxicity), and malignant hyperthermia. In order to clinically differentiate between these three conditions, examination of deep tendon reflexes is recommended. In serotonin syndrome, brisk to exaggerated reflexes can be found with the lower limb reflexes being more exaggerated as compared to the upper limbs. The presence of ankle clonus and opsoclonus has been also described in serotonin syndrome with increasing toxicity. One point to remember in severe serotonin syndrome is that hypertonicity can mask the typical and diagnostic indicators of hyperreflexia and clonus. Another overdose that is associated with brisk to exaggerated reflexes is sympathomimetic overdose.

Neuroleptic malignant syndrome will have a history of recent intake of a neuroleptic drug or a dopamine antagonist and will have autonomic instability and extrapyramidal effects. Malignant hyperthermia occurs following exposure to trigger agents, such as volatile anesthetics or depolarizing muscle relaxants, and is associated with mixed respiratory and metabolic acidosis and hyperkalemia.

Respiratory Examination

The key aspects to examine are the respiratory rate and the amount of secretions.

In general, it will be good to ask for the respiratory rate at admission to the ED as this could help in toxidrome identification. Bradypnea can occur either due to depression of the CNS due to overdose with opioid, sedative, alcohol or antiepileptics, or respiratory muscle failure due to neuromuscular blocking agents, OP poisoning, or snake envenomation. Tachypnea is usually related to CNS stimulants (sympathomimetics, central hallucinogens, and drug withdrawal states). It can also be a compensatory response to either hypoxia (tissue hypoxia due to agents such as carbon monoxide, cyanide, and methemoglobinemia or hypoxemia due to respiratory irritants or cardiotoxic agents) or metabolic acidosis due to methanol, ethanol, ethylene glycol, or iron overdose.

It is also advisable to find out how much secretions the patient had at admission to the hospital. Assessment of the need for frequent suctioning in the absence of fever or purulent secretions can help one suspect a cholinergic crisis as seen with OP poisoning.

Cardiovascular System Examination

Further clues in the examination that could facilitate narrowing down the diagnosis in the hyperthermic patient include *assessment of the heart rate and BP*. Whilst sympathomimetic syndrome and anticholinergic and serotonin syndrome typically present with hypertension and tachycardia, NMS typically has autonomic instability with labile BP and heart rate changes. If the patient does not have diaphoresis, this can help narrow down the diagnosis to a possible anticholinergic syndrome.

In the situation, where a poisoned patient presents with hypertension and bradycardia, possible common agents would include overdose with alpha-adrenergic agonists and clonidine. Hypotension and bradycardia are generally associated with cholinergic overdose (OP and carbamates)

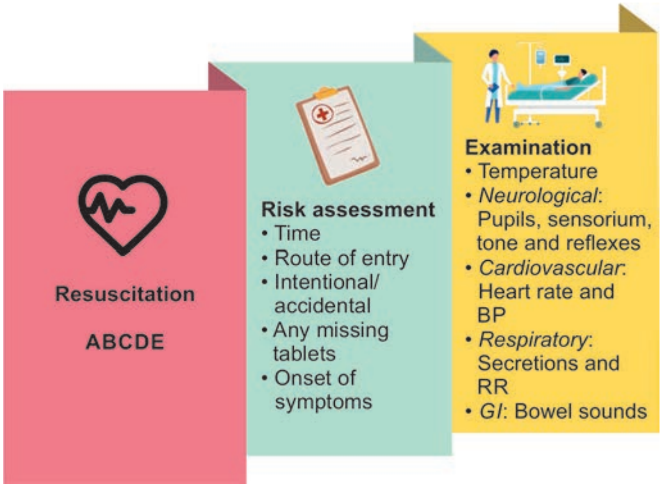
Resuscitation
ABCDE
Risk assessment
• Time
• Route of entry
• Intentional/ accidental
• Any missing tablets
• Onset of symptoms
Examination
• Temperature
• *Neurological*: Pupils, sensorium, tone and reflexes
• *Cardiovascular*: Heart rate and BP
• *Respiratory*: Secretions and RR
• *GI*: Bowel sounds

Fig. 1: Clinical approach to unknown poisoning. (ABCDE: airway, breathing and circulation, disability, and exposure; BP: blood pressure; GI: gastrointestinal; RR: respiratory rate)

or overdose with beta blockers, calcium channel blockers, digoxin, opioids, and antiarrhythmic agents.

Abdomen Examination

This generally comes in handy in a patient with mydriasis. Exaggerated bowel sounds in a *hyperthermic patient with mydriasis* are seen with sympathomimetic and serotonin syndromes. Anticholinergic syndrome, on the other hand, causes decreased bowel sounds. Cholinergic syndrome will also present with exaggerated bowel sounds—such patients will have miotic pupils.

Putting It All Together

A flowchart showing the abovedescribed approach has been made to facilitate a quick assessment of a poisoned patient **(Fig. 1 and Table 2)**. The current case scenario, there was a history of polypharmacy overdose with the patient having predominant clinical features of a serotonin syndrome. The presence of normal pupils is most likely due to the combination of drugs consumed.

Pitfalls of Clinical Examination in a Poisoned Patient

It is important to keep in mind the numerous obstacles that prevent effective diagnosis of the acute toxidrome. Inaccurate ingestion history, a short or lengthy time delay between exposure and physical examination, or the impact of the patient's usual cardiovascular drugs in concealing the predicted physiologic response may all contribute to discrepancies between the history and the physical examination. Additionally, overlapping characteristics are seen in many toxidromes and patients may not exhibit all the symptoms or evidence associated with a particular toxidrome. Tricyclic antidepressants may cause miosis, despite being anticholinergic, due to competing effects from activation of muscarinic and alpha receptors. If a patient has a combined overdose (e.g., heroin and methamphetamine), the patient's pupils are likely to be between pinpoint and dilate.

Individual variation, comorbid diseases, and medications can also influence the clinical findings. As an illustration, a patient receiving beta-adrenergic receptor antagonists may not have the tachycardia typically seen with sympathomimetic or anticholinergic toxidromes. Additionally, there may some outliers within each group that may lack toxidrome findings in some cases, as is seen with opioid analgesics, such as meperidine, which does not have the characteristic opioid toxicity syndrome.

SUMMARY

Before proceeding with identifying the toxidrome, urgent supportive treatment and monitoring of vital signs must be done for individuals who arrive with severe toxic symptoms or after potentially fatal ingestions. The clinical evaluation and differential diagnosis must always exclude alternative explanations. Recognizing the poison, either through a patient's history, the identification of a toxidrome, or laboratory investigation, can help steer care and patient disposition. There are various life-saving antidotes available, and patients who may benefit from them should be identified as quickly as possible.

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Chapter

30

Examination of a Critically Ill Obstetric Patient

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ABSTRACT

Although obstetric patients form only 1–2% of admissions to a general intensive care unit (ICU), they present a unique challenge to the intensivist. Disorders requiring critical care differ in each trimester of pregnancy and in postpartum patients. Maternal early warning criteria are often used to identify patient with critical illness. History of problems in previous pregnancies should be obtained as many disorders may recur in subsequent pregnancies. Common symptoms are fever, abdominal pain, vomiting, cough, shortness of breath, headache, blurring of vision, vaginal bleeding, and urinary frequency. Specific symptoms should be sought based on the suspected diagnosis after the initial history. As a result of physiological changes in pregnancy, many "abnormal" vital parameters and physical findings may actually be normal in obstetric patients. The most important signs to look for on general examination in pregnant women are pallor, edema, rash and petechiae, and ecchymosis. Systemic examination should be aimed at identifying diagnostic signs and severity of organ dysfunction or failure. After 24 weeks of pregnancy, unstable patients should be in the supine position for the least possible time when examining to avoid aortocaval compression, which can worsen hypotension. Detailed obstetric assessment should be performed and fetal monitoring is important in later stages of pregnancy using bedside cardiotocography. Point-of-care ultrasonography at the bedside is an extremely useful adjunct to physical examination for make a diagnosis and to monitor these patients. Optimal patient management involves early involvement of a multidisciplinary team consisting of an obstetrician, intensivist, neonatologist, and anesthesiologist.

INTRODUCTION

Obstetric patients include women admitted during pregnancy or the first 42 days (6 weeks) after delivery or termination of pregnancy. Although obstetric patients form only 1–2% of admissions to a general intensive care unit (ICU), they present a unique challenge to the intensivist because of the physiological changes associated with pregnancy and puerperium, the specific diseases peculiar to pregnancy, and the need to take care of both the mother and the fetus. While pregnant women (antepartum) may get admitted, often near full term, with obstetric or medical disorders, postpartum women comprise approximately 60–80% of obstetric admissions to the ICU.

An intensivist must be familiar with common problems that are encountered in obstetric patients in the ICU. Critical illness in obstetric patients fall into one of four categories:

1. *Obstetric disorders (unique to pregnancy):* Ruptured ectopic pregnancy, placental abruption, placenta previa, postpartum hemorrhage (PPH), adherent placenta, severe pre-eclampsia, hypertensive crisis, eclampsia, HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, uterine perforation during abortion or uterine rupture, tocolytic-induced pulmonary edema, and ovarian hyperstimulation syndrome (OHSS)
2. *Medical disorders that are aggravated by pregnancy:* Valvular heart disease, congenital cyanotic heart disease, primary pulmonary hypertension, obstructive sleep apnea, chronic kidney disease, prolactinoma, diabetes mellitus, cirrhosis, sickle cell disease, scleroderma, systemic lupus erythematosus, intracranial tumors, and multiple sclerosis
3. *Medical disorders that tend to be more severe during pregnancy:* Infections such as urinary tract infection, listeriosis, viral hepatitis E, *Plasmodium falciparum* malaria, varicella pneumonia, influenza A (H1N1) infection, disseminated intravascular coagulation (DIC), venous thromboembolism, postpartum

hemolytic–uremic syndrome (HUS)/thrombotic thrombocytopenic purpura (TTP), Sheehan syndrome, aspiration of gastric contents, and acute respiratory distress syndrome (ARDS)

4. *Disorders unrelated to pregnancy:* Toxoplasmosis, community-acquired pneumonia, drug abuse or overdose, trauma

CASE SCENARIO

A 28-year-old primigravida in the 34th week of pregnancy was admitted to the ICU with symptoms of worsening edema of the legs and severe headache since 3 days. She had been detected to have hypertension in the 26th week of pregnancy and was on oral labetalol 100 mg three times a day, on which the blood pressure was well controlled. On enquiry, she also complained of pain in the right hypochondrium since 1 day prior to admission, Physical examination revealed a heart rate of 114 beats/min, with blood pressure (BP) 160/104 mm Hg, respiratory rate (RR) 20 breaths/min and mildly raised jugular venous pressure (JVP). SpO_2 *was 94% on room air. She had pitting edema of both feet and ankles, mild icterus, and no fever.*

Systemic examination showed that there was no murmur or gallop, breath sounds were normal, and there were no rales or wheeze. The abdomen was distended and the uterus was enlarged to 4 cm below the xiphisternum. There was tenderness in the right hypochondrium although liver and spleen were not palpable. She was alert, but confused and answered only a few questions correctly. Vision was normal and she had no focal neurological signs or neck rigidity.

A diagnosis of severe pre-eclampsia with mildly deranged liver function tests and mild thrombocytopenia was made. The obstetrician ordered electronic fetal monitoring with cardiotocography. Appropriate treatment was started and after appropriate treatment (antihypertensives, intravenous magnesium sulfate infusion, intravenous dexamethasone for fetal lung maturity, and laboratory assessment of renal, liver, coagulation, and urinary parameters). It was decided to carry out urgent delivery by cesarean section.

After cesarean delivery, the premature neonate was transferred to the neonatal ICU and the mother was back in the adult ICU. Three hours postdelivery, the nurse informed that the patient was having vaginal bleeding. The obstetrician found on examination that the uterus was flabby and not contracted. A diagnosis of atonic PPH was made. Appropriate treatment was started. Two hours later, she also developed coffee grounds nasogastric tube aspirate and thrombo-elastography showed features of disseminated intravascular coagulopathy.

Assessment of a Critically Ill Obstetric Patient

As in all critically ill patients, initial assessment is to assess the airway, breathing, and circulation and to start immediate resuscitation measures to stabilize these if they are severely compromised. After this, one can go on to the formal clinical examination to make a specific diagnosis and to identify all the organ systems that will need intensive support.

The purpose of examination of a critically ill obstetric patient is to:

- Make a diagnosis of the acute problem—obstetric or medical disorder?
- Detailed assessment of organ dysfunction/failure
- Continued targeted monitoring to detect clinical deterioration or improvement
- Obstetric assessment of the pregnancy and fetal well-being
- For planning delivery if needed, and optimization of the patient for urgent delivery

Stage of Pregnancy

An important first step in the assessment of an obstetric patient is to determine the duration of pregnancy or time since delivery. Specific problems unique to various stages of pregnancy **(Table 1)**.

The Maternal Early Warning Criteria are used to identify pregnant women who need to be considered for admission to an ICU. These criteria are recalibrated to account for these physiological changes and are listed in **Table 2**.

Obtaining a Relevant History

History of previous pregnancies and any problems in previous pregnancies should be obtained. Some obstetrics disorders may recur in subsequent pregnancies such as systemic lupus erythematosus (SLE), antiphospholipid syndrome, pre-eclampsia, peripartum cardiomyopathy, gestational diabetes mellitus, placenta previa, abruptio placentae, pregnancy-related liver disorders, preterm premature rupture of membranes, spontaneous preterm delivery, and stillbirth have a tendency to recur in the subsequent pregnancy.

TABLE 1: Problems seen during specific stages of pregnancy and postpartum period.

Stage of pregnancy	*Disorders seen*
First trimester	• Hyperemesis gravidarum • Ruptured ectopic pregnancy • Septic abortion • Ovarian hyperstimulation syndrome
Second trimester	• Septic abortion • Thromboembolic disorders
Third trimester	• Severe pre-eclampsia/eclampsia • HELLP syndrome • Acute fatty liver of pregnancy • PRES • Thromboembolic disorders • Antepartum hemorrhage: – Placenta previa – Placental abruption – Vasa previa • DIC
Postpartum period—first 24 hours	• All conditions listed under third trimester • Eclampsia • Postpartum hemorrhage • Obstetric shock • Retained placenta • Uterine inversion • Amniotic fluid embolism
Postpartum period—after 24 hours	• Cerebral venous sinus thrombosis • Thromboembolic complications • Puerperal sepsis • Breast abscess

(DIC: disseminated intravascular coagulation; HELLP: hemolysis, elevated liver enzymes, low platelet count; PRES: posterior reversible encephalopathy syndrome)

The next step is to get a detailed history of the various symptoms. Common symptoms are fever, abdominal pain, vomiting, cough, shortness of breath, headache, blurring of vision, vaginal bleeding, and urinary frequency.

Common causes of these symptoms in pregnant women are shown in **Table 3**. Specific symptoms should be sought based on the suspected diagnosis after the initial history.

Specific symptoms should then be sought based on the suspected diagnosis after the initial history.

Physical Examination

Initial examination as in other ICU patients consists of vital signs followed by a systematic examinations of various organ systems. This is not easy in pregnant women because physiological changes in pregnancy may alter physical signs. Some signs that would be abnormal in nonpregnant patients could be normal in pregnancy, for example, tachycardia, tachypnoea, raised JVP, or mild pedal edema. Common physiological changes in pregnancy and how they can affect interpretation of physical and bedside monitor parameters are shown in **Table 4**.

TABLE 2: Maternal Early Warning criteria as per the guidelines for obstetric high-dependency unit (HDU) and intensive care unit (ICU) (Government of India, 2016).

	Critical values	
Parameters	*For HDU admission*	*For ICU admission*
Systolic blood pressure (BP)	<90 and >160 mm Hg	<80 mm Hg, or 30 mm Hg below patient's usual BP
Diastolic blood pressure	<50 and >110 mm Hg	
Heart rate	>110 and <60 beats/min	<50 and >140 beats/min
Respiratory rate	>25 breaths/min	<8 and >35 breaths/min
SpO_2	<95%	
Urine output	>0.5 mL/kg/h (>30 mL/h)	<400 mL in 24 hours, or <160 mL in 8 hours
Level of consciousness		Unarousable patient

General Examination

Besides the vital signs, most important signs to look for in pregnant women are pallor, edema, rash and petechiae, and ecchymosis.

Edema in the first trimester can be a sign of OHSS. It can also be seen in normally some women late pregnancy. Edema can be due to hypoproteinemia due to pre-eclampsia or chronic liver disease, or due to congestive heart failure. Unilateral edema can be a sign of deep vein thrombosis, which is common in pregnancy. Rash is often due to allergy, exanthematous infections. It is important to diagnose the infection causing rash due to the possible fetal consequences of some infections such as rubella, cytomegalovirus (CMV), toxoplasmosis, or acute human immunodeficiency virus (HIV) infection. Petechiae, ecchymosis, and purpura may be due to thrombocytopenia seen in obstetric conditions such as pre-eclampsia, HELLP syndrome, DIC, TTP, or medical

TABLE 3: Common symptoms in pregnancy and their causes.

Symptoms	***Common causes in pregnancy***
Fever	Infections—urinary tract infection, respiratory infection/pneumonia, uterine infections (septic abortion, chorioamnionitis, and puerperal sepsis), tropical infections, viral hepatitis
Abdominal pain	Gastritis, cholecystitis, ruptured ectopic pregnancy, labor pain, and placental abruption
Vomiting	Hyperemesis gravidarum, severe preeclampsia, acute fatty liver of pregnancy, gastroenteritis, and viral hepatitis
Cough	Respiratory infection, acute pulmonary edema (valvular heart disease, peripartum cardiomyopathy), and aspiration of gastric contents
Shortness of breath	Asthma, cardiac disorders, pulmonary thromboembolism, respiratory infections, ARDS, and amniotic fluid embolism
Headache	Severe preeclampsia, PRES, intracranial hemorrhage, cerebral venous thrombosis, and intracranial infections
Blurring of vision	Severe preeclampsia, PRES, and raised intracranial pressure
Vaginal bleeding	Antepartum hemorrhage and postpartum hemorrhage
Urinary frequency	Physiological in later part of pregnancy, urinary tract infection, polyuria in severe diabetes

(ARDS: acute respiratory distress syndrome; PRES: posterior reversible encephalopathy syndrome)

TABLE 4: Physiological changes in pregnancy and how they affect assessment of a critically ill patient.

Physiological changes	***Clinical consequences***
Cardiovascular changes: • Increased blood volume • Increased cardiac output • Decreased systemic vascular resistance • Decreased colloid oncotic pressure • Supine aortocaval compression	• Dilutional anemia • Heart rate increased by 15–20 beats/min, cardiac murmurs • BP decreases by 10–15 mm Hg • Susceptible to third spacing of fluid • Can decrease cardiac output by 30% after 24 weeks of pregnancy
Respiratory: • Increased minute ventilation • Raised diaphragm near full term • Increased oxygen consumption by 20%	• Increase respiratory rate, tidal volume, decreased $PaCO_2$, respiratory alkalosis, compensatory decreased in serum bicarbonate (reduced buffering capacity) • Reduced functional residual capacity • More prone to hypoxia
GI changes: • Decreased GI motility • Relaxation of lower esophageal sphincter	• Constipation, increased gastric emptying time • Increased risk of aspiration of gastric contents
Uterus: • Uterine blood flow = 10% of cardiac output • Aortocaval compression in supine position • Poor autoregulation of uterine blood flow	• Decreased blood flow to vital organs in shock • Supine hypotension syndrome, difficulty in CPR, need to keep patient in lateral position in ICU • Prone to hypoperfusion and fetal distress in shock
Acid-base physiology: • Effect of progesterone on respiration—hyperventilation	• $PaCO_2$ decreased to 26–30 mm Hg • Serum bicarbonate decreased (compensated respiratory alkalosis) to 17–18 mMol/L

(BP: blood pressure; CPR: cardiopulmonary resuscitation; GI: gastrointestinal; ICU: intensive care unit)

disorders such as immune thrombocytopenia (ITP), SLE, and malaria. Ecchymosis may be seen in coagulopathic conditions such as liver involvement in pre-eclampsia, acute fatty live of pregnancy or due to DIC. It can also occur in medical disorders such as acute viral hepatitis E infection or leptospirosis.

Systemic Examination

As in other ICU patients, systemic examination should be comprehensive and aimed at identifying diagnostic signs and also presence of, and severity of, organ dysfunction or failure. It is extremely important to keep unstable patients in the supine position for the least possible time when examining to avoid aortocaval compression after 24 weeks of pregnancy, which can cause hypotension and hemodynamic compromise.

Cardiovascular Examination

Hypertension is seen in pre-eclampsia, eclampsia, SLE, and raised intracranial pressure. Hypotension may be due to hypovolemia, hemorrhage, sepsis, cardiogenic shock due to valvular or congenital heart disease, peripartum cardiomyopathy, or obstructive shock due to pulmonary thromboembolism. Hypotensive patients beyond 24 weeks of pregnancy should be immediately placed in a lateral position. Bounding pulses and systolic murmurs are physiological while diastolic murmurs are usually pathological.

Central venous pressure (CVP) may be falsely elevated in advanced pregnancy. Bedside 2D echocardiography is important to identify the cause of hypotension and shock. Inferior vena cava (IVC) diameter and collapsibility are not reliable in pregnancy due to raised intra-abdominal pressure.

Respiratory System

Tachypnea is common in normal pregnancy. Look for use of accessory respiratory muscles and signs of impending respiratory arrest like fatigue, drowsy, labored breathing, silent chest, bradycardia, cyanosis, and inability to complete sentences. Auscultation may reveal signs of consolidation. Decreased breath sounds at the bases are common near full-term pregnancy due to raised domes of the diaphragm. Pulmonary edema may be evident with crackles and wheeze will be heard in exacerbation of asthma.

Interpretation of arterial blood gas (ABG) should take into account the low $PaCO_2$ and bicarbonate that are physiological in pregnancy. Point-of-care ultrasound (POCUS) is important because of the need to avoid unnecessary chest X-rays in pregnancy. It can be used to detect pneumonia, pneumothorax, pleural effusions, pulmonary edema, and right ventricular dysfunction in pulmonary thromboembolism.

Neurological Examination

Consciousness can be assessed by Glasgow Coma Scale (GCS). Any GCS <15 should arouse suspicion of severe pre-eclampsia, hepatic encephalopathy, posterior reversible encephalopathy syndrome (PRES), TTP, cerebral venous sinus thrombosis, stroke, or intracranial infection.

Localizing signs are indicative of stroke or intracranial hemorrhage, whereas areflexia is a sign of magnesium toxicity in patients receiving magnesium sulfate for pre-eclampsia/eclampsia. Neck rigidity suggests meningitis or subarachnoid hemorrhage.

Abdominal Examination

Abdominal examination is challenging in pregnancy due to the presence of the pregnant uterus. Palpation of the liver, spleen, and kidneys can be difficult and use of POCUS is extremely helpful as an adjunct to physical examination of the abdomen.

Liver dysfunction may manifest as jaundice, asterixis, edema, ascites, petechiae, and ecchymosis due to coagulopathy.

Renal dysfunction will result in oliguria, edema, puffiness of the face, and ascites in pre-eclampsia. Renal angle tenderness is seen in pyelonephritis. Encephalopathy and myoclonic jerks may be a sign of uremia in acute kidney injury.

Gastrointestinal dysfunction may be seen in the form of paralytic ileus or signs of acute pancreatitis or cholecystitis. Paralytic ileus should prompt a search for an acute abdominal condition—ruptured ectopic pregnancy, perforative peritonitis, uterine sepsis, intra-abdominal hemorrhage, or acute pancreatitis before attributing it to metabolic causes. Ascites in the first trimester can be a sign of ovarian hyperstimulation syndrome.

Obstetric Evaluation

The obstetrician plays a key role in taking care of the pregnancy. However, an intensivist must know some basic points about obstetric examination.

- *Points to note in history are:*
    - *Gravida:*____________________ *Para:* ____________
      *Living:*____________________ *Abortion:* _________
      Last menstrual period: _________ *Expected due date:* ___________ *Gestational age:*_______ *weeks*
- *Examination of a pregnant patient:*
    - *Fundal height*________________
    - *Presentation:* Vertex/Breech/Transverse/Other ________________________
    - *Uterine tenderness:* Yes/No
    - *Fetal heart rate:* Present/Absent, if yes______/min
    - *Amniotic fluid:* Clear/Blood stained/Meconium stained

- *Examination of a postpartum patient:*
  - *Labor:* Spontaneous/induced labor/prelabor cesarean section
  - Course of labor: Uneventful/prolonged 1st stage/prolonged 2nd stage/obstructed
  - Mode of delivery: Spontaneous vaginal delivery/assisted vaginal delivery/cesarean section/miscarriage
  - Physical examination
  - Uterus—contracted, atonic, perforation/rupture, inversion
  - Abdomen—cesarean section scar—clean, infected, tenderness, hematoma
  - Perineum, vagina and cervix for injury, laceration, hematoma, and infection
  - PPH: Yes/no
  - Vaginal discharge: Normal/foul smelling
  - Breast examination: Normal/engorged/abscess

Focused Disease-specific Assessment

Acute abdomen:

- The differential diagnosis is extensive, including any cause of abdominal pain, such as acute gastroenteritis, peptic ulcer disease, small bowel obstruction, inflammatory bowel disease, acute pancreatitis, ectopic pregnancy, ruptured ovarian cyst, ovarian torsion, renal colic, pyelonephritis, and (rarely) such disorders as sickle cell crisis, diabetic ketoacidosis, and porphyria.
- A thorough physical examination is vital in localizing the site of pain and tenderness. POCUS is also an important diagnostic tool especially in liver or biliary disorders, hydronephrosis due to ureteric calculus, ruptured ectopic pregnancy, and ovarian pathology.

Ruptured ectopic pregnancy:

- An ectopic pregnancy is an extrauterine pregnancy, most commonly occurring in the fallopian tube.
- The classic presentation of ectopic pregnancy is vaginal bleeding and/or abdominal pain in the setting of a positive pregnancy test in the first trimester of pregnancy, usually 6–8 weeks after the last menstrual period.
- The cause of bleeding should be evaluated by transvaginal ultrasound.
- Rupture of an ectopic pregnancy can result in life-threatening intra-abdominal hemorrhage and the patient presents with severe or persistent abdominal pain or symptoms suggestive of ongoing blood loss. Feeling faint, syncope, or loss of consciousness due to hemorrhagic shock is common. The history of a missed menstrual period may not be available in some patients.
- If there is sufficient intra-abdominal bleeding to reach the diaphragm, referred pain may be felt in the shoulder.
- Blood pooling in the posterior cul-de-sac (pouch of Douglas) may cause an urge to defecate.
- POCUS is diagnostic in these patients.

Ovarian hyperstimulation syndrome:

- OHSS is caused by controlled ovarian stimulation with gonadotropins for treatment of infertility.
- The already high gonadotrophic effect present before conception increases further during pregnancy due to increase in endogenous human chorionic gonadotrophin that enhances the effect of vascular endothelial growth factor.
- There is a resultant increase in vascular permeability and transudation of fluid causing edema, bilateral pleural effusion, and ascites.
- Hemoconcentration and blood hyperviscosity may result in thromboembolic events such as pulmonary thromboembolism or thrombotic stroke.
- OHSS can present with abdominal pain and bloating, nausea, and vomiting.
- POCUS will reveal enlarged ovaries, ascites, and pleural effusions of varying severity.
- In severe cases oliguria, thromboembolism, and ARDS may be seen.

Pre-eclampsia/eclampsia:

- Pre-eclampsia patients may be asymptomatic and may deteriorate rapidly without any specific symptoms and signs. Early clinical feature include blood pressure ≥140/90 mm Hg, edema feet, and mild proteinuria, which develop after 20 weeks of gestation.
- Severe pre-eclampsia is characterized by development of organ damage and dysfunction. Early symptoms include headache, visual blurring or visual loss, confusion, oliguria, and increasing edema; many of these manifestations were seen in the case discussed above.
- PRES should be considered in the setting of pre-eclampsia with vision loss or deficit, headache, and altered sensorium or confusion.
- Seizures indicate the development of eclampsia, but can also occur with severe hypertension leading to intracranial hemorrhage.

- Hyperreflexia and severe headache are common features of impending eclampsia.
- Right hypochondrial pain is indicative of liver disease and one should rule out subcapsular liver hematoma.
- Edema of the extremities is a common finding.

Antepartum hemorrhage:

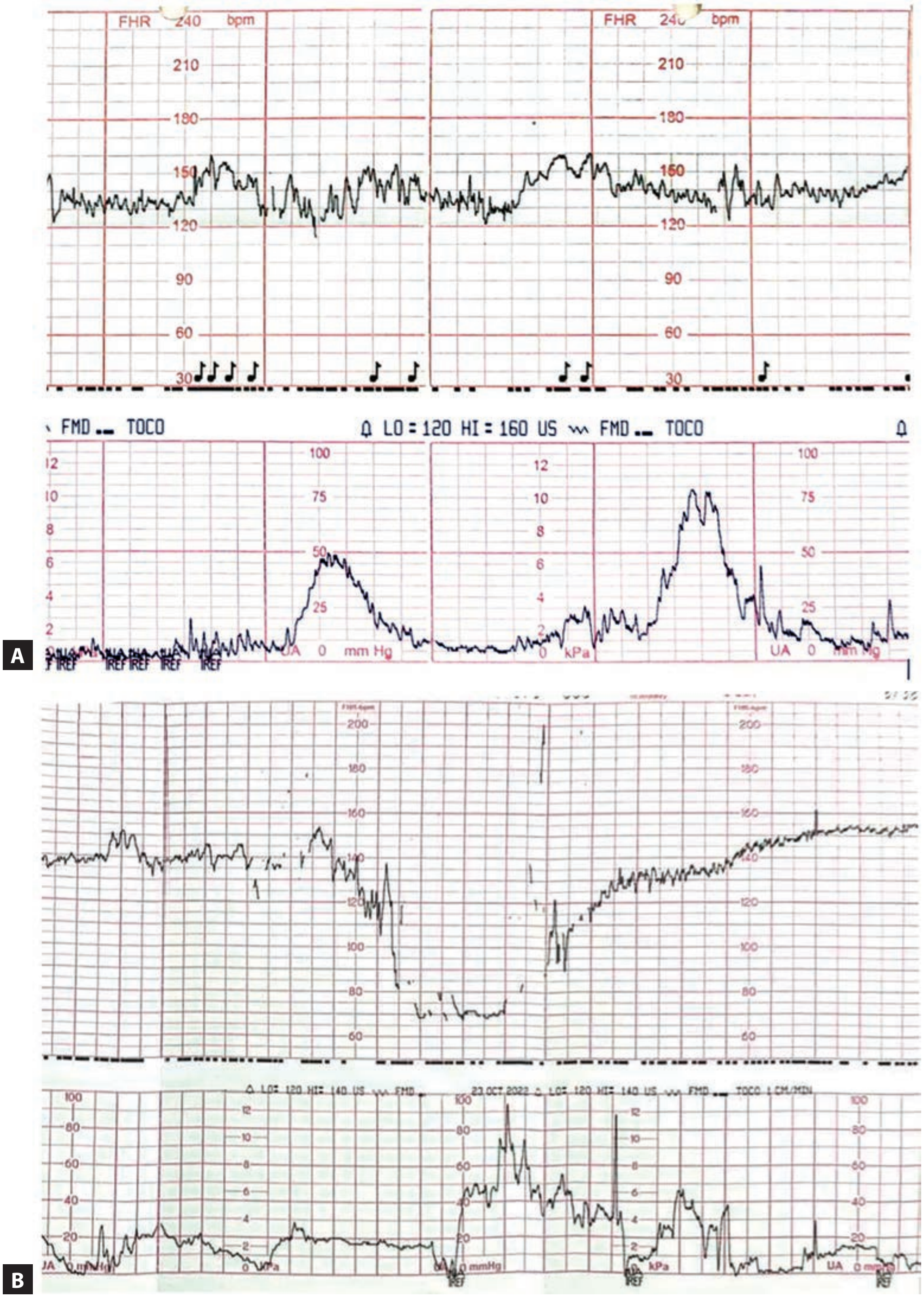
- Antepartum hemorrhage commonly occurs due to placental abruption, and sometimes due to placenta previa. Other causes include vasa previa (where the blood is from the fetus) and genital tract lesions (cervical polyps or erosions) where the bleeding is of maternal origin.
- *Abruptio placentae:*
    - In a woman with antepartum hemorrhage, pelvic examination should be avoided until placenta previa is excluded on ultrasound.
    - Placental abruption refers to premature separation of a normally implanted placenta from the uterine wall. The bleeding that results may be revealed vaginally (in 80% of cases) or concealed within the uterus (20%). Abruption that is clinically recognized complicates 1% of all pregnancies. The diagnosis is confirmed by ultrasonography.
    - Risk factors should be looked for pre-eclampsia, prior placental abruption, blunt trauma, smoking, cocaine abuse, uterine anomaly or fibroids, multiparity, advanced maternal age, preterm premature rupture of membranes, chorioamnionitis, and thrombophilia.
    - Signs and symptoms of placental abruption may include vaginal bleeding (80%), uterine tenderness or back pain (65%), and nonreassuring fetal heart rate monitoring (60%). Other findings include frequent, hypertonic uterine contractions, and preterm labor. Uterine tenderness suggests extravasation of blood into the myometrium (couvelaire uterus).
    - The amount of vaginal bleeding is not a reliable indicator of the severity of the hemorrhage since bleeding may be concealed.
    - It is often associated with DIC because of release of prothrombotic products from the retroplacental clot and due to consumption of clotting factors and may cause PPH. Atonic PPH may also occur following delivery.
- *Placenta previa:*
    - Placenta previa refers to a placenta that overlies or is proximate to the internal os of the cervix. It complicates 0.3–0.5% of pregnancies and the majority of cases are diagnosed during routine sonography in asymptomatic women, usually during the second trimester.
    - Most women with placenta previa do not develop symptoms during pregnancy. However, some women may present with acute onset of bright red vaginal bleeding, which is usually painless. Bleeding may sometimes be profuse and result in antepartum hemorrhage and shock.

Postpartum hemorrhage:

- Traditionally, PPH has been defined as blood loss >500 mL with vaginal delivery or >1,000 mL with cesarean delivery. This was redefined by the American College of Obstetrics and Gynecology in 2017, as cumulative blood loss >1,000 mL with signs and symptoms of hypovolemia within 24 hours of the birth process, regardless of the route of delivery.
- Primary PPH is a bleeding that occurs in the first 24 hours after delivery, whereas secondary PPH is bleeding 24 hours to 12 weeks postpartum.
- There are four main causes of postpartum hemorrhage that account for the majority of cases. Also known as the "Four T's", these are tone (uterine atony), tissue (retained placenta), trauma (laceration), and thrombin (coagulopathy).
- Tone—uterine atony, or lack of effective contraction of the uterus, is the most common cause of PPH. PPH in a previous pregnancy is a significant risk factor.
- Tissue refers to retained placenta, which is commonly due to severe degree of placenta accreta, where the placenta invades the myometrium or surrounding tissues. This should be diagnosed early and treatment is usually hysterectomy.
- Trauma—laceration in the perineum, vagina, or cervix during vaginal delivery can result in persistent PPH and should be diagnosed by physical examination and appropriately repaired. Deep tears may extend beyond the genital tract and can result in retroperitoneal bleeding.
- Thrombin refers to coexisting coagulation problems such as thrombocytopenia (pre-eclampsia or HELLP syndrome), deficiency of clotting factors (acute fatty liver of pregnancy), or DIC [intrauterine fetal death (IUFD), placental abruption, severe pre-eclampsia, and amniotic fluid embolism] that often complicate obstetric disorders. These could also occur in medical

disorders complicating pregnancy such as acute viral hepatitis or dengue infection. Physical examination may reveal petechiae, ecchymosis, and bleeding from other mucosal or cutaneous sites in patients with coagulation defects or thrombocytopenia.

- Another often missed diagnosis in PPH is uterine inversion. A soft, "boggy" or noncontracted uterus is the common finding with uterine atony. Uterine inversion presents as a round bulge or mass with palpation of the fundal wall in the cervix or lower uterine segment



Figs. 1A and B

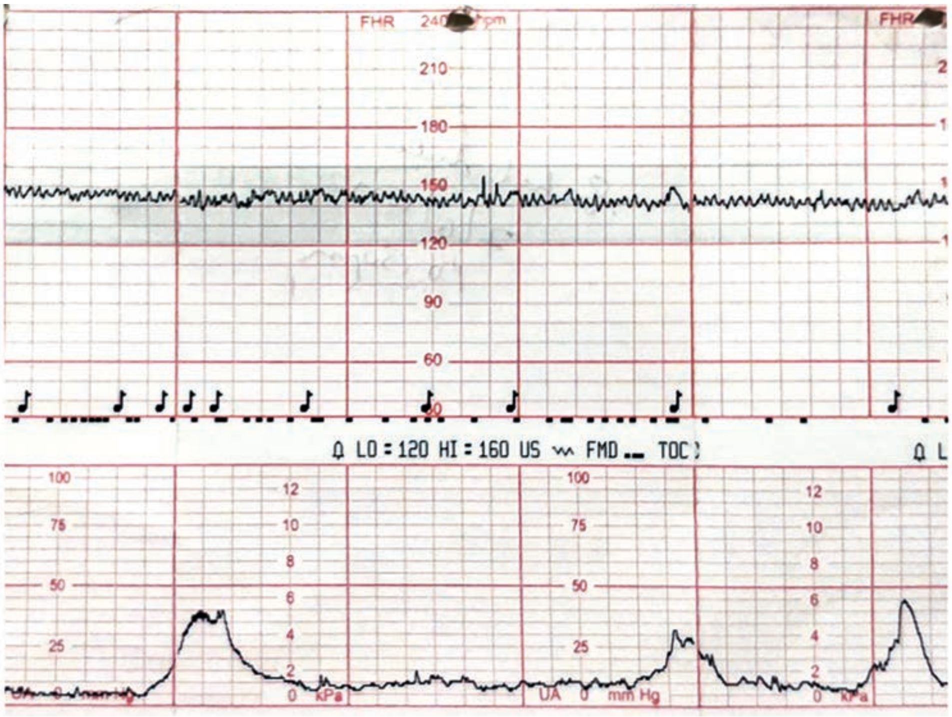


Fig. 1C

Figs. 1A to C: Cardiotocogram—each tracing has 2 parts—the upper tracing is the fetal heart rate (FHR) and lower tracing is the uterine contraction (tocogram). (A) A normal tracing—fetal movements are shown by musical symbols at the bottom of the FHR tracing. Normally, fetal movements are accompanied by a small increase in FHR, while uterine contraction is not accompanied with any change in heart rate; (B) Deceleration of FHR during the uterine contraction indicating fetal compromise; (C) Absence of increase in FHR with fetal movements, which is an abnormal response. There is also no change in heart rate with uterine contraction, which is a normal response. This is a nonreassuring tracing and needs further assessment (e.g. by biophysical profile assessment) to confirm if there is fetal compromise or not.

and is often associated with excessive traction on the umbilical cord or abnormally adherent placenta.

- Secondary causes of PPH include retained products of conception, infection, subinvolution of the placental site, and inherited coagulation deficits.

Fetal Monitoring in the Intensive Care Unit—The American College of Obstetricians and Gynecologists

Fetal monitoring is an important part of monitoring of a critically ill pregnant patient in the ICU. Continuous electronic fetal heart rate monitoring may show changes in the fetal heart rate tracing due to decreased uteroplacental perfusion and fetal acid–base status.

These can be corrected by optimizing maternal blood pressure, ventilation, acid–base balance, or cardiac output. If the changes persist, this could be an indication for urgent delivery.

More intensive monitoring can be done at the bedside by cardiotocography, which measures changes in fetal heart rate and its relationship to fetal activity (nonstress test or NST) and during uterine contractions (contraction stress test or CST) **(Figs. 1A to C)**. Fetal heart rate is measured by a Doppler ultrasound transducer placed on the abdomen and uterine contractions are measured by an external tocodynamometer attached to the mother's abdomen. A number of fetal heart rate parameters including baseline rate, heart rate variability, and accelerations and decelerations during uterine contractions are measured and findings are interpretated as reassuring or nonreassuring. A nonreasssuring observation may prompt more detailed assessment of fetal wellbeing by doing the biophysical profile (BPP), which combines fetal NST along with ultrasonography to assess of amniotic fluid volume, fetal tone, fetal breathing, and spontaneous fetal movement.

SUMMARY

- Critical illness in obstetric patients may be due to obstetric or medical disorders.
- Different disorders are common in different stage of pregnancy.
- As a result of physiological changes in pregnancy, many "abnormal" vital parameters and physical findings may actually be normal in obstetric patients.
- Use of early warning scores can be used to recognize clinical deterioration.
- Clinical examination should be directed toward diagnosis of the causative disorder, identification of organ dysfunction, fetal well-being, and preparing targets for parameters to be optimized.
- An intensivist must be well versed in eliciting a specific history and performing physical examination of a critically ill pregnant or postpartum woman.
- Point of care ultrasonography at the bedside is an extremely useful adjunct to physical examination to make a diagnosis and to monitor these patients.
- After making a diagnosis, attention to specific symptoms and signs is the key to detect complications early.
- Optimal patient management involves early involvement of a multidisciplinary team consisting of an obstetrician, intensivist, neonatologist, and anesthesiologist.

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Chapter 31

How will I Examine a Patient with Weaning Failure?

Anirban Bhattacharjee, Rajeshwari Subramaniam

ABSTRACT

This chapter focuses on the assessment and management of patients who have failed weaning from mechanical ventilation. It touches upon the primary causes that contribute to weaning failure, followed by a systematic, case-based approach to tackle weaning failure.

INTRODUCTION

Although mechanical ventilation is an important intervention for most critically ill patients in the intensive care unit (ICU), unnecessary prolonged mechanical ventilation is associated with various issues such as diaphragmatic atrophy, venous thromboembolism, delirium, and pneumonia. Therefore, it is crucial to establish a strategy to reduce the duration of mechanical ventilation and restore independent breathing as soon as invasive mechanical ventilation is initiated. On the other hand, prematurely discontinuing ventilation can impose additional stress on the respiratory and cardiovascular systems. Successful liberation from mechanical ventilation requires a customized plan for each patient, because weaning failure, subsequent postextubation distress, reintubation attempts, and resumption of mechanical ventilation are linked to significant morbidity and increased mortality. It has been estimated that medical patients spend approximately 40–50% of their total time on mechanical ventilation in the weaning process (Boles et al., 2007).

Generally, approximately 80% of patients receiving ventilatory support are successfully extubated at the first attempt and are subsequently transferred to a step-down facility. However, around 20–30% of patients fail the first spontaneous breathing trials (SBTs) (Beduneau et al., 2017; Esteban et al., 1997; Heunks & Van Der Hoeven, 2010). Patients categorized as having "difficult weaning" require up to three SBTs or up to 1 week to achieve liberation from ventilation. On the other hand, patients with "prolonged weaning" have failed their third SBT or have been unable to be extubated for more than a week since the first SBT.

Weaning failure is defined as the inability to tolerate an SBT or the need for reintubation within the first 2 days after a successful SBT and extubation. Weaning difficulties commonly arise in patients with chronic obstructive pulmonary disease (COPD), restrictive pulmonary disease, heart failure, and neuromuscular disorders.

CASE SCENARIOS

1. *Mr AD, a 75-year-old male patient, was admitted to the ICU with pneumonia and acute exacerbation of COPD. Initially, he underwent a short trial of noninvasive ventilation (NIV), but due to worsening respiratory status, he required intubation. He received treatment with antibiotics, vasopressor infusions, and steroids to manage his condition. 3 days later, an attempt was made to conduct an SBT, but it had to be discontinued due to respiratory distress.*
2. *Mrs F, a 40-year-old female patient, was admitted to ICU after laparotomy for perforation and peritonitis. Her ICU course was complicated by abdominal sepsis, septic shock, acute respiratory distress syndrome (ARDS), and multiorgan failure, necessitating vasopressors, upscaling of antibiotics, neuromuscular blockade, steroids, and renal replacement therapy (RRT). 2 weeks after admission she was hemodynamically stable and considered for SBT, which she failed repeatedly.*

Clinical Approach to Weaning Failure

History

A clinician should be acquainted with the clinical background of the patient to be able to diagnose and manage the cause of weaning failure.

- *Demographic factors:* Elderly patients with age >65 years are at higher risk of weaning difficulty due to cardiorespiratory comorbidities. Females are at higher risk of developing postextubation stridor and extubation failure, and therefore, a cuff leak test is advisable, more so in case of prolonged ventilation >7 days or repeated intubations.
- *Nutritional status:* Malnutrition is commonly seen in critically ill patients and is seen to be associated with worse outcomes. Nutritional evaluation with validated scores such as NUTRIC score should be done in critically ill patients.
- *Past and present medical history:* The history of the patient reveals the existence and severity of chronic cardiopulmonary illnesses that put patients at high risk of weaning failure, such as COPD, ischemic heart disease (IHD), and chronic heart failure. In any case of difficult weaning after resolution of acute illness and the presence of adequate respiratory function, underlying cardiovascular dysfunction should be sought after. History of dyspnea and its severity, angina on exertion or rest and its severity, effort tolerance, paroxysmal nocturnal dyspnea, and orthopnea can be useful in diagnosing chronic cardiac dysfunction. History of neurological disorders, hypothyroidism, and medication history of use of steroids should be noted to identify a high risk of weaning failure.

Physical Examination of a Patient with Weaning Failure

General examination: A focused general clinical examination of a patient with weaning failure should include:

- The presence of pallor can identify severe anemia that may impede weaning by placing a demand load on the cardiorespiratory system.
- Peripheral edema and distended neck veins suggest circulatory overload or cardiac failure, and warrant optimization of fluid balance and cardiac medications.
- Fluid balance: Intake and output charting should be reviewed. Weight gain is an important indicator of fluid overload, which may hamper weaning.
- Anthropometric measurements are often unreliable in intensive care setup and therefore, a proper history of nutritional intake and use of scoring systems such as NUTRIC or NRS-2002 scores are relied upon.
- The presence of fever may indicate nonresolution of the initial infectious process or new hospital-acquired infection.

Systemic examination: Respiratory system: The presence of tachypnea with a rapid and shallow pattern of breathing can indicate impending weaning failure. Auscultation of the chest can provide important clues regarding the underlying cause of weaning failure.

If localized bronchial breath sounds or adventitious sounds such as crackles and rhonchi are heard during chest auscultation, it suggests pneumonic consolidation is a possible cause. Bilateral crackles may indicate pulmonary edema or ARDS. Bronchospasm, characterized by wheezing or, in severe cases, a silent chest, can also contribute to weaning failure. Treatment with bronchodilators, with or without corticosteroids, may be necessary in such cases.

In situations where tracheal narrowing, tracheomalacia, or laryngeal edema is suspected as the cause of extubation failure, a cuff leak test should be performed before the next extubation attempt. A formal endoscopic evaluation of the airway is very useful after prolonged tracheal intubation to diagnose tracheomalacia, stenosis, mucosal edema, and hypertrophy.

Cardiovascular system: Although patients with COPD or preexisting heart disease are at an increased risk of developing weaning-induced pulmonary edema (WiPO), it can also occur in patients without prior cardiac or pulmonary disease. Clinical findings suggestive of WiPO include crepitations on lung auscultation, the presence of a third heart sound, elevated jugular venous pressure, hepatojugular reflex, and peripheral edema. It is important to take note of the cumulative fluid balance in patients who fail weaning, as fluid overload can contribute to the development of WiPO.

Neurological: The patient's consciousness and level of sedation can be assessed using the Glasgow Coma Scale (GCS) and the Richmond Agitation Sedation Scale (RASS). These tools help evaluate the patient's neurological status and the depth of sedation. A sedation protocol aimed at minimizing sedation has been recommended to expedite liberation from mechanical ventilation (Ouellette et al., 2017).

Delirium screening is essential in the ICU, and the Confusion Assessment Method for the ICU (CAM-ICU) is commonly used for this purpose. The presence of delirium is an independent predictor of a prolonged duration of mechanical ventilation (Shehabi et al., 2010).

Musculoskeletal: To assess for ICU-acquired weakness (ICUAW), the muscle strength of the patient can be evaluated using the Medical Research Council (MRC) scale. This helps in determining if weakness or muscle dysfunction is contributing to difficulties in weaning from mechanical ventilation.

In neurologically impaired patients, it is important to assess swallowing function to rule out the risk of aspiration after extubation. Swallowing assessments, such as bedside evaluations or formal swallowing studies, can help determine the patient's ability to safely swallow and prevent complications postextubation.

Adjuncts to Clinical Examination

Blood gas analysis: Arterial blood gas gives important information regarding the gas exchange performance of the lungs and the presence of shunt or dead space ventilation, and information about the oxygen supply and demand of the body. Elevation of lactate and decrease in central venous saturation of oxygen may indicate the inability of the heart to meet the increased load on the cardiorespiratory system imposed by unassisted breathing.

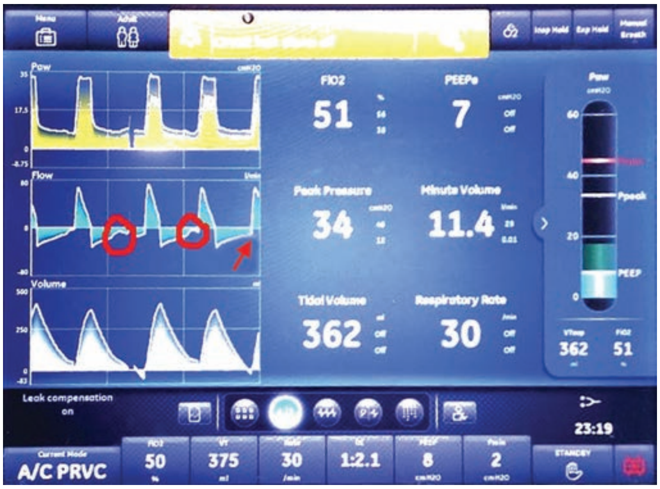
Mechanical ventilatory data: Pressure–time scalars and pressure–volume loops provide a qualitative assessment of airway resistance and compliance. An examination of scalar waveforms can unravel the underlying pathophysiology of air trapping and dynamic hyperinflation. Patient–ventilator dyssynchrony can increase the patient's work of breathing and cause SBT failure **(Fig. 1)**. A quantitative estimate can be done by measuring peak pressure (P_{peak}) and plateau pressure (P_{plat}) in volume controlled and flow-limited ventilation, by the formula:

$$R_{aw} = \frac{P_{peak} - P_{plateau}}{Flow}$$

The value is normally about 5–7 $cmH_2O/L/sec$ in a ventilated patient. The possible causes of increased airway resistance and peak airway pressures include bronchoconstriction, airway secretions, and kinking or block of the endotracheal tube **(Table 1)**.

Compliance is usually reduced in patients with ARDS who fail weaning. It can be calculated as follows:

$$C_{static} = \frac{V_t}{P_{plateau} - PEEP}$$



FIO2 51 %
PEEPe 7 cmH2O
Peak Pressure 34 cmH2O
Minute Volume 11.4 l/min
Tidal Volume 362 ml
Respiratory Rate 30 /min
VTexp 362 ml
FIO2 51 %
Current Mode A/C PRVC
FIO2 50 %
VT 375 ml
Rate 30 /min
I:E 1:2.1
PEEP 8 cmH2O
Pmin 2 cmH2O

Fig. 1: Ventilator waveform showing air-trapping (arrow) and consequent ineffective trigger asynchrony (circles) due to increased inspiratory load because of intrinsic positive end-expiratory pressure (PEEP).

TABLE 1: Factors affecting respiratory mechanics.

Increased airway resistance	• Small diameter of endotracheal tubes • Tube obstruction or kinking • Airway secretions • Bronchoconstriction • Peribronchiolar edema • Tracheomalacia or tracheal stenosis
Decreased respiratory system compliance	*Reduced lung compliance:* • Pneumonia • Pulmonary edema • ARDS • Interstitial lung disease and lung fibrosis • Intrinsic PEEP *Reduced chest wall compliance:* • Obesity • Chest wall edema • Intra-abdominal hypertension and abdominal compartment syndrome • Pleural fluid and pneumothorax • Ascites

(ARDS: acute respiratory distress syndrome; PEEP: positive end-expiratory pressure)

The disease conditions associated with reduced compliance of respiratory system include pneumonia, ARDS, pulmonary edema, abdominal and chest wall edema, obesity, pleural effusion, and ascites.

Ultrasonography: Lung and diaphragm ultrasound: Lung ultrasound is an ingenious method of using reverberation artifacts to diagnose abnormalities in lung. A normally aerated lung does not allow the ultrasound beam to penetrate beyond the visceral pleural leading to horizontal equidistant mirror artifacts parallel to the pleura called A-lines. The visceral pleural slides on the parietal pleural if there is no air or fluid separating the two structures. Therefore, a positive lung slide and A-lines suggest a normally aerated lung. The presence of lung sliding rules out pneumothorax. Similar findings can be seen if respiratory failure is due to severe bronchospasm, as in severe asthma or COPD.

The presence of fluid in lung interstitium and alveoli leads to vertical bright line artefacts extending from the pleural line down to the end of the ultrasound image. Increasing severity of edema leads to an increasing number of B lines until they coalesce in alveolar edema. More severe de-aeration, as in consolidation and collapse, can lead to the appearance of the lung as tissue (tissue sign). The regularity of pleura and distribution of B lines can give clues to differentiating between cardiogenic and noncardiogenic pulmonary edema. Cardiogenic pulmonary edema usually appears as bilaterally present, symmetrical B lines with regular pleural lines. Inflammatory pulmonary edema is often asymmetrical, unilateral, and associated with pleural irregularities or breaks at the margin of consolidated and normal lungs, called shred sign.

Another noninvasive method of assessing diaphragm strength and integrity is by using ultrasound. The parameters commonly measured by ultrasound are diaphragmatic thickness, thickening fraction (TF), and diaphragmatic excursion (DE) as shown in **Figures 2A and B**. The linear ultrasound probe is placed between anterior and midaxillary line at 8th–9th intercostal space to visualize the zone of apposition of diaphragm. The diaphragm is seen as a hypoechoic muscular layer between the hyperechoic pleura and peritoneum (shown with an asterixis in **Figs. 2A and B**). End-inspiratory (T_{insp}) and end-expiratory (T_{exp}) thicknesses are measured in M-mode and TF is calculated as:

$$TF = \frac{T_{insp} - T_{exp}}{T_{exp}} \times 100$$

In order to measure DE, the curvilinear probe is placed in the subcostal area between the midclavicular and anterior axillary line, directed cranially, medially, and posteriorly. The view uses acoustic windows of liver or spleen to visualize the right or left hemidiaphragm. Visualization of the left hemidiaphragm is often problematic due to the smaller acoustic window provided by the spleen. The diaphragm appears as a curved hyperechoic line excursing toward and away from the probe during inspiration and expiration. An M-mode line is positioned perpendicular to the excursion of diaphragm to measure the DE. DE <1.0 cm and TF <20–30% are often taken as cut-offs to predict weaning failure due to diaphragmatic dysfunction.

Echocardiography: Echocardiographic monitoring of patients before and during weaning can unravel the diagnosis of cardiac dysfunction. Important clues are:

- Increased E velocity and decreased velocity of mitral annular plane (E′)
- Increased E/E′
- New or worsening regional wall motion abnormalities may suggest myocardial ischemia during weaning.
- Increase in lung ultrasound scores during weaning.

Laboratory investigations: Severe anemia can impede weaning by placing an additional load on the cardiorespiratory system. Dyselectrolytemia can lead to respiratory muscle weakness and difficult weaning. Severe hypothyroidism and adrenal insufficiency are also rare causes of weaning failure and should be looked for and treated **(Table 2)**.

Figure 3 is an illustration of the general approach to the evaluation of weaning failure.

Pathophysiology of Weaning Failure

There are five important determinants of weaning outcome (Heunks & Van Der Hoeven, 2010):

1. "Brain dysfunction" or the central drive
2. Airway and lung dysfunction
3. Cardiac dysfunction
4. Diaphragmatic dysfunction
5. Endocrine/metabolic derangement.

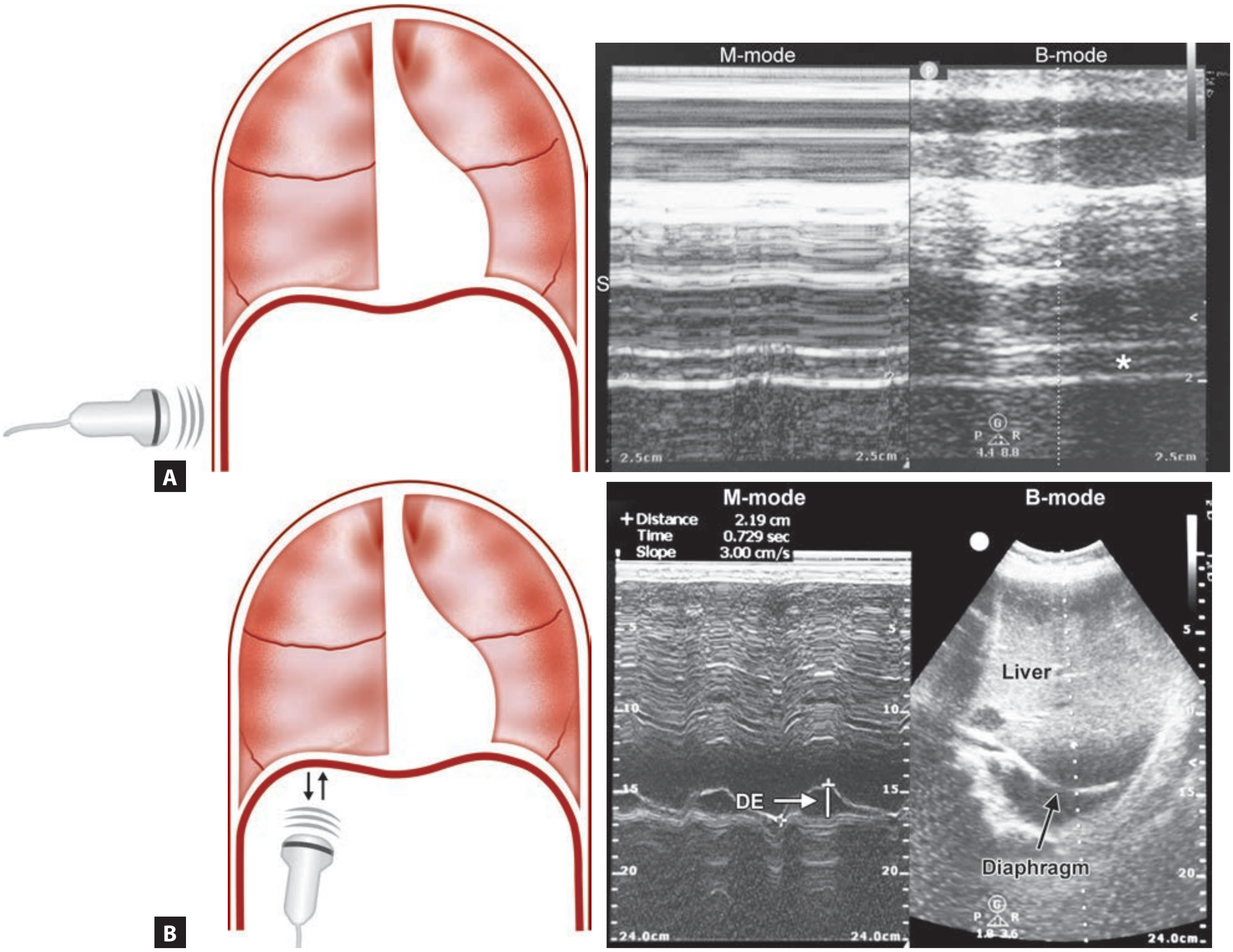
Imbalance in any of these, singly or in combination, may impede successful weaning.

Brain Dysfunction

Impaired cognitive function has been seen to be associated with a more than fourfold higher risk of failed extubation. Anxiety and depression may also contribute to the failure of weaning. Central drive may be reduced by excess sedation or neurological diseases and lead to more time on mechanical ventilation.

Airway and Lung Dysfunction

Failure of the patient's respiratory mechanics ("pump") can occur due to increased resistive or elastic load, resulting in weaning failure. Poor gas exchange due to


M-mode
B-mode
S
2.5cm
2.5cm
2.5cm
*
A
M-mode
B-mode
+ Distance 2.19 cm
Time 0.729 sec
Slope 3.00 cm/s
Liver
DE
Diaphragm
24.0cm
24.0cm
24.0cm
B

Figs. 2A and B: Ultrasonography of diaphragm. (DE: diaphragmatic excursion)

TABLE 2: Diagnosis of common causes of weaning failure.

	History	*Clinical examination*	*Investigations*
Weaning-induced pulmonary edema	• History of dyspnea on exertion, angina pectoris, orthopnea, and paroxysmal nocturnal dyspnea • Comorbidities such as COPD and ischemic heart disease	• Distended neck veins and raised JVP • Pedal edema • Bilateral fine crackles • S3 gallop	• Bilateral fluffy opacity in chest X-ray (Bat wing appearance) • Bilateral symmetrical B lines with regular pleura • Bilateral pleural effusion • Distended IVC • Elevated CVP • Poor LV ejection fraction • Regional wall motion abnormalities • Diastolic dysfunction • Elevated NTproBNP levels • Low $ScvO_2$ during SBT • Increase in hematocrit during SBT

Contd…

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	History	***Clinical examination***	***Investigations***
Lung parenchymal disease	• Diagnosis of pneumonia or ARDS • History of productive cough, fever, and increased endotracheal secretions	• Fever • Increased secretions, purulent character of secretions • Bronchial breath sounds • Adventitious sounds—crackles and rhonchi	• Poor PaO_2:FiO_2 ratio • High plateau pressures and reduced compliance of respiratory system. • Lobar or interstitial opacities in chest X-rays • B or B′ profile in lung ultrasound, presence of shred sign, tissue sign, parapneumonic effusion • Normal echocardiography
Obstructive airway diseases	• History of COPD and bronchial asthma • Smoking history	• Cachexia and barrel chest • Use of accessory muscles of respiration • Wheeze or silent chest on auscultation	• Hyperinflated lung field on chest Xray. Concomitant pneumonia can also be seen • High peak airway pressure and airway resistance, normal plateau pressure with widened gap between peak and plateau pressures • A profile with normal DVT scan • Features of cor pulmonale in echocardiography • Respiratory acidosis or post-hypercapneic metabolic alkalosis in ABG, both of which can impede weaning
Diaphragmatic dysfunction	• Prolonged mechanical ventilation • Known neuromuscular diseases • Development of ICU-acquired weakness • History of use of neuromuscular blockers and corticosteroid	• Patients do well on assisted mode of ventilation but have rapid shallow breathing during SBT • ICU-acquired weakness (MRC score <48)	• Reduced Maximum inspiratory pressure • Ineffective trigger asynchronies • Diaphragmatic thickening fraction less than 20% • Diaphragmatic excursion less than 11 mm
Neurological abnormality	• Stroke and head injury • History of dementia, substance abuse, etc.	• Poor GCS or FOUR score • Delirium as per CAM-ICU score • RASS score not in target range • Swallowing abnormality	• Computerized tomography of brain • Fiberoptic endoscopic evaluation of swallowing (FEES)
Intra-abdominal issue	History of abdominal trauma, complicated intra-abdominal infections, and ascites	• Abdominal distension • Elevated upper margin of liver dullness • Reduced air entry in basal auscultation zones	• Raised intra-abdominal pressure • Ascites and pleural effusions in imaging

(ARDS: acute respiratory distress syndrome; CAM: Confusion Assessment Method; COPD: chronic obstructive pulmonary disease; FOUR: Full Outline of Unresponsiveness; ICU: intensive care unit; JVP: jugular venous pressure; SBT: spontaneous breathing trial; GCS: Glasgow Coma Scale)

ventilation–perfusion (V/Q) mismatch or shunt, the inability to protect the airway and clear secretions, airway obstruction, and/or stridor, either individually or in combination, are responsible for the majority of instances of weaning failure.

In some cases, upper airway obstruction may arise due to tracheal stenosis or the development of granulations when mechanical ventilation exceeds 4 weeks. The presence of an endotracheal tube itself contributes to significant resistance during spontaneous breathing, and

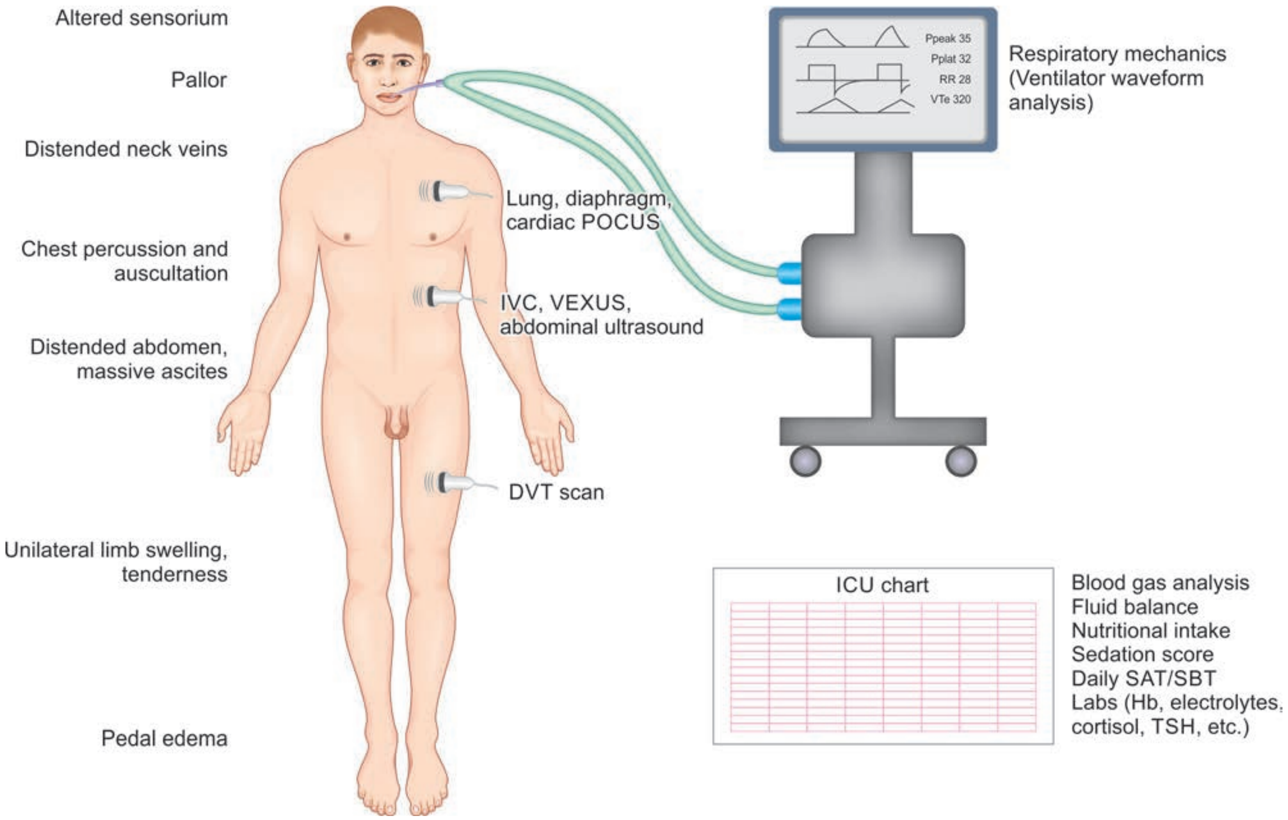
Altered sensorium
Pallor
Distended neck veins
Chest percussion and auscultation
Distended abdomen, massive ascites
Unilateral limb swelling, tenderness
Pedal edema
Lung, diaphragm, cardiac POCUS
IVC, VEXUS, abdominal ultrasound
DVT scan
Ppeak 35
Pplat 32
RR 28
VTe 320
Respiratory mechanics (Ventilator waveform analysis)
ICU chart
Blood gas analysis
Fluid balance
Nutritional intake
Sedation score
Daily SAT/SBT
Labs (Hb, electrolytes, cortisol, TSH, etc.)

Fig. 3: Evaluation of a patient with weaning failure. (DVT: deep vein thrombosis; ICU: intensive care unit; POCUS: point-of-care ultrasound; SAT: spontaneous awakening trial; SBT: spontaneous breathing trial; TSH: thyroid-stimulating hormone)

its removal can lead to airway obstruction due to edema. Patients who fail weaning trials often exhibit higher airway resistance.

In patients with COPD, intrinsic positive end-expiratory pressure (PEEPi) can impede weaning efforts. PEEPi develops due to increased flow resistance, expiratory flow limitation, high breathing frequency, and loss of elastic recoil of the lung. PEEPi acts as an inspiratory threshold, increasing the work of breathing. Pulmonary hyperinflation caused by PEEPi places the diaphragm at a suboptimal position on the length-tension curve, impairing its ability to generate the required negative pressure. Additionally, PEEPi contributes to ineffective triggering and patient–ventilator asynchrony, ultimately leading to respiratory fatigue.

An ominous pattern characterized by “rapid and shallow” breathing serves as an indicator of a failing respiratory pump. This pattern is characterized by a decrease in tidal volume (Vt), accompanied by an increase in respiratory rate. As the Vt decreases, the dead space to Vt ratio increases, resulting in reduced alveolar ventilation. This can have detrimental effects on overall respiratory function.

Cardiac Dysfunction

Weaning from mechanical ventilation can potentially trigger latent heart failure and lead to a condition called weaning-induced pulmonary edema through various mechanisms. Cardiac dysfunction is observed in a significant percentage (44–87%) of weaning failures (Santangelo et al., 2022). While patients with preexisting heart disease or COPD are at a higher risk of developing WiPO, it can also occur in patients without prior cardiac or pulmonary issues.

The primary initiating factor for WiPO is the changes in intrathoracic pressure during the weaning process. Several mechanisms have been proposed to explain this phenomenon.

- *Redistribution of venous blood:* Increased adrenergic tone during weaning can cause the redistribution of venous blood from the unstressed volume to the stressed volume. This leads to an elevation in systemic venous pressure gradient, central blood volume, and right ventricular (RV) preload.

- The shift from positive intrathoracic pressure during mechanical ventilation to negative pleural pressure during weaning results in an increase in left ventricular (LV) preload, afterload, systolic arterial pressure, and heart rate. This can elevate LV end-diastolic pressure, contributing to the development of pulmonary and bronchial wall edema. As a result, the work of breathing increases.

Echocardiographic studies have shown that WiPO is most commonly associated with LV diastolic dysfunction (Routsi et al., 2019). The elevated LV end-diastolic pressure during weaning can induce pulmonary edema and lead to increased respiratory effort.

Diaphragmatic Dysfunction

Inappropriate ventilatory settings, drug administration (such as steroids and neuromuscular blockers), and electrolyte imbalances are common causes of ventilator-induced diaphragmatic dysfunction (VIDD) and/or ICUAW, both of which contribute to difficulties in weaning from mechanical ventilation. VIDD is defined as the “loss of force-generating capacity of the diaphragm specifically related to the use of mechanical ventilation” and is characterized by disuse atrophy and microstructural changes. VIDD can begin as early as 12 hours after the initiation of controlled mechanical ventilation.

Animal studies have demonstrated that the development of diaphragmatic dysfunction is influenced by the ventilatory strategy (Gayan-Ramirez et al., 2005; Sassoon et al., 2004). Continuous mandatory ventilation has been associated with atrophy of both type I and type IIb/x fibers of the diaphragm, while intermittent spontaneous breathing helps preserve diaphragm fiber dimensions and proportions (Gayan-Ramirez et al., 2005).

A systematic review of 31 studies revealed that ICUAW is seen in 43% (interquartile range of 25–75%) of ICU patients (Fan et al., 2014). Both VIDD and ICUAW are associated with high severity of illness, multiorgan failure, hyperglycemia, and the use of drugs such as steroids and neuromuscular blockers (Vanhorebeek et al., 2020).

Metabolic Factors Contributing to Weaning Failure

- Malnutrition in ICU leads to decreased muscle mass and difficult weaning. Clinicians should ensure that adequate nourishment with both macro- and micronutrients is provided as per existing guidelines.
- Hypokalemia, hypomagnesemia, and hypophosphatemia can lead to respiratory muscle weakness and prevent successful weaning.
- In rare cases, adrenal insufficiency and hypothyroidism have been found to be associated with weaning failure. Cortisol and thyroid hormone supplementation may help in weaning such patients.

CASE SCENARIOS REVISITED

1. *Mr AD, a 75-year-old male patient, was admitted to ICU with pneumonia and acute exacerbation of COPD. After a brief trial of NIV he was intubated and treated with antibiotics, vasopressor infusions, and steroid. 3 days later he underwent an SBT which had to be terminated due to respiratory distress.*

   Physical examination revealed a poor nutritional status as often seen in patients with COPD. His blood pressure readings were high most times of the day. On auscultation, crackles were heard in bilateral infra-axillary, interscapular, and infrascapular areas. Lung ultrasonography revealed a bilateral B profile in lateral and posterior zones, with minimal bilateral pleural effusion. Tissue sign and shred were seen in the right posterolateral zone. Echocardiography revealed normal systolic function and grade 2 diastolic dysfunction. Laboratory investigations revealed high serum NTpro-BNP (N-terminal pro b-type natriuretic peptide) levels. A review of fluid balance revealed a cumulative fluid balance of 4 L. The patient was started on diuretics and antihypertensive medications were reviewed. Improvement in lung aeration was seen with serial ultrasonography. After improvement of his blood gas and acceptable respiratory status, he was extubated to NIV support and observed for extubation failure. The patient was maintained on conservative fluid management and nocturnal NIV on discharge from ICU.
2. *Mrs F, a 40-year-old female patient, was admitted to ICU after laparotomy for perforation and peritonitis. Her ICU course was complicated by abdominal sepsis, septic shock, ARDS, and multiorgan failure, necessitating vasopressors, upscaling of antibiotics, neuromuscular blockade, steroids, and RRT. 2 weeks after admission she was hemodynamically stable and considered for SBT, which she failed repeatedly.*

   On examination, she was conscious, obeying commands, and hemodynamically stable. She was not pale or edematous. Her oxygen saturation

(SpO_2) was 90% and her respiratory rate was 15 breaths/min on assist/control mode of ventilation with expired tidal volume (VTe) 400 mL, PEEP 5 cmH_2O, and fraction of inspired oxygen (FiO_2) 40%. Ppeak was 20 cmH_2O, Pplat 19 cmH_2O, and C_{RS} 28 mL/cmH_2O. She had a few crackles on auscultation in both infrascapular zones. Her muscle power was reduced with an MRC score of 36.

On lung ultrasound, she had bilateral A profile anteriorly, A profile with scattered B lines <3 per ISS laterally, and consolidation at posterior-most regions. Diaphragm ultrasound during SBT showed a TF of 15%. A clinical diagnosis of ICUAW was made with diaphragmatic dysfunction being the cause of weaning failure. A muscle biopsy showed features of necrotizing myopathy. Creatine phosphokinase (CPK) levels were mildly elevated. Patient's nutrition and electrolyte status were reviewed and hypokalemia and hypomagnesemia were corrected. Patient's glycemic control was reviewed, and steroid was tapered off. Daily spontaneous awakening and breathing trial, mobilization, and physiotherapy were done. Patient was tracheostomized after failing two SBTs, which led to a further reduction in need for sedation. Patient was eventually weaned to T-piece oxygen supplementation and was decannulated after 1 month of stay in high-dependency unit (HDU).

SUMMARY

Weaning a patient from mechanical ventilation requires optimization of the respiratory, cardiac, and metabolic status of the patient while preserving the function of the respiratory pump. A plan for weaning should be made immediately after intubation and weaning should start as soon as acute respiratory failure improves. A systematic assessment of patients is needed to find out and treat the factors impeding weaning. The clinical examination should be supplemented with ultrasonography, evaluation of respiratory mechanics from ventilator waveforms, blood gas, and relevant investigations to precisely address the cause of weaning failure.

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Chapter

32

How will I Approach and Examine a Patient with Delirium?

Kushal Kalvit, Atul Prabhakar Kulkarni

ABSTRACT

Delirium is a very common and an often overlooked condition in critically ill patients. Although it is easier to recognize hyperactive delirium, the more common mixed subtype and also the hypoactive subtype goes undiagnosed. A systematic approach is necessary in the evaluation of delirium so as to arrive at a cause and treat accordingly. Prevention and early recognition of delirium reduces morbidity and improves outcomes in intensive care unit (ICU) patients.

INTRODUCTION

With the advent of increased screening tools and awareness, delirium is being increasingly recognized in all intensive care units (ICUs). However, it remains neglected as most caregivers are focused only on treating the disease for which the patient has been admitted. Delirium in the ICU can present as hyperactive, mixed, or hypoactive form. The hyperactive form is the most easily recognized form and is associated with less mortality. However, the hypoactive/mixed forms often go unnoticed, which leads to an increased risk of mortality. Management of delirium starts with a systematic screening, assessment for etiology, and multifaceted treatment of all the causative factors.

CASE SCENARIO 1

A 70-year-old man was admitted with fever, cough, and shortness of breath. He had a history of hypertension, diabetes mellitus, and transient ischemic attack (TIA). He was taking telmisartan, hydrochlorothiazide, metformin, aspirin, and atorvastatin for the comorbidities. He was shifted to ICU in view of progressive dyspnea. He was started on antibiotics and was put on high-flow nasal oxygenation (HFNO). His hypoxia improved and he was hemodynamically stable for the next 2 days. On the third day, the nurse called to report that the patient appeared confused and had an irrelevant talk. The intensivist noticed that the patient was calmly sitting in the bed but was not paying any attention to what he was being asked.

Nomenclature

An acute change in the baseline mental state of a patient is variably described by intensivists all over the world. Terms such as altered sensorium, altered mental state, acute confusional state, and encephalopathy are loosely and interchangeably used to describe the same set of symptoms. This often leads to miscommunication between caregivers and may lead to inappropriate management. In order to standardize the nomenclature and improve communication, an updated consensus statement by 10 societies [including European Society of Intensive Care Medicine (ESICM) and Society of Critical Care Medicine (SCCM)] was released in 2020. The following are the salient features of the same:

- The term "acute encephalopathy" is the preferred term for any acute (usually <4 weeks) change in baseline cognitive status. Acute encephalopathy can have a range of presentations from subtle subsyndromal delirium, delirium to coma, and can also include physical manifestations such as seizures or extrapyramidal signs.
- The terms "altered mental status", "acute confusional state", "acute brain failure", and "acute brain dysfunction" should *not* be used instead of or in addition to acute encephalopathy.
- The term delirium has to be used only if the patient fulfills the DSM-5 (Diagnostic and Statistical Manual-5) criteria. This criterion states that delirium is an acute and fluctuating change from the baseline level of attention and awareness in addition to a change in

cognition. This change should not be because of a preexisting neurocognitive deficit *and* should be explained by another medical condition, drug/toxin exposure or withdrawal. Additionally, the change in mental status should *not* be because of severely depressed consciousness.

- The term "subsyndromal delirium" is to be used for patients who have symptoms of delirium but do not fulfil the entire DSM-5 criteria.
- The term "coma" is to be used for severely depressed consciousness as per the Glasgow Coma Score (GCS) or Full Outline of Unresponsiveness (FOUR) score.

Quick Assessment

The initial few minutes are crucial in order to identify and treat the life-threatening causes of an acute change in mental state. The search for various clues and management of the same goes hand in hand. The most important causes to rule-in or rule-out at this stage are airway obstruction, hypoxia due to respiratory failure, hypoglycemia, shock with or without hypotension, hypertensive emergency, stroke (ischemic or hemorrhagic), tachyarrhythmias, and bradyarrhythmias.

While attending to such a scenario in the ICU, the following schema is helpful for most of the patients. The following steps are a rough guide and need to be modified on a case-by-case basis.

1. Take a quick look at the monitor that is attached to the patient. A glimpse reveals abundant information regarding the vital signs. The oxygen saturation (SpO_2) value and waveform, heart rate, rhythm, and blood pressure are the parameters one must take note of. Once noted, one needs to start treating the abnormal parameters in addition to further examination. Hypoxia, hypotension, hypertensive emergency, and arrhythmias need to be managed with oxygen supplementation, fluids/vasopressors, antihypertensives, and antiarrhythmics, respectively.
2. Airway and breathing assessment should be done together. Airway needs to be assessed to rule out obstruction. One can start by simply observing the pattern of patient's breathing. An acutely obstructed airway often leads to a very anxious patient who gasps for air frantically. Ask the patient his/her name or any other simple question and listen to the voice. A loud and clear voice indicates no impending obstruction although it may change over time, depending on the underlying pathology.
3. Breathing is initially assessed by noting the respiratory rate, use of accessory muscles of respiration, symmetric movement of the chest, ability of the patient to speak in sentences and auscultation for wheeze (bronchospasm), crepitations (pulmonary edema), or absent breath sounds (pneumothorax, status asthmaticus, pleural effusion, and hemothorax).
4. Palpate and compare all four limbs as the presence of cold extremities is an early sign of shock. One should always remember that shock can be present despite a normal blood pressure and heart rate and needs to be corrected early.
5. A quick neurological examination needs to be done to identify stroke. GCS, pupillary abnormalities, movement of all limbs, asymmetry of face, neck stiffness, dysphasia, and ophthalmoplegia are the most important points to be examined.
6. While doing all the abovementioned assessment, one can ask the bedside nurse or assistant to perform a fingerstick glucose testing to identify hypoglycemia or hyperglycemia. Temperature should be measured as certain neurological conditions are associated with hyperthermia.

Relevant History

Once the initial assessment is done and the urgent issues are addressed, one needs to thoroughly review the case and look for additional clues that have caused or may contribute to the acute encephalopathy.

- *Past medical history:*
  - Comorbidities:
    - For example, diabetes mellitus, hypertension, coronary artery disease, chronic respiratory disorders, thyroid disorders, and malignancy
    - Anticipate the possible direct complications that may arise due to them.
    - A previously well-controlled condition may get exacerbated due to the current illness leading to encephalopathy.
  - Medications:
    - Enquire about current medications in the hospital as well as prior medications at home (including nonprescription drugs, over-the-counter medicines, and alternative medicines)
    - Look for newly added medications, recent change in the dosage, abrupt stopping, or irregular compliance with the prescription.

    - Drug–drug interactions also need to be considered.
  - Recent procedures/surgery that the patient has underwent.
  - Alcohol abuse and illicit drug use
  - Recent/past vaccinations
- *Details of the present illness:*
  - To be taken from the caregivers at home as the patient may or may not be in a position to answer the questions.
  - Onset and temporal progression of the current illness that has led to hospitalization
  - Recent fever and travel history
  - Accurately elicit the baseline neurocognitive state of the patient from the caregivers. Ask specific questions and enlist the physical/mental tasks that the patient could or could not do independently. It is advisable to document and communicate the same with other clinicians to avoid vague descriptions.
  - Severity of illness for which the patient was admitted and the length of stay in the hospital/ICU. These factors themselves may trigger symptoms suggestive of delirium.

A commonly ignored point at this stage is the presence of communication barrier due to different languages spoken by the patient and clinician. Make every effort to communicate with the patient in his/her language with the help of caregivers or translators. A patient speaking in different language is often termed as “uncooperative” or “altered sensorium” mistakenly! Lastly, one should always ask if the patient uses any visual or hearing aids and that they need to be installed immediately.

How to Examine a Patient with Delirium?

Focused General Physical Examination

- Inspect the scalp and face for signs of trauma such as a scalp hematoma, bruising behind the pinna, clear fluid leaking from the pinna or nose, and raccoon eyes. The trauma may be the indication for hospitalization or may happen in the hospital due to fall or self-inflicted injury in a combative patient.
- Inspect the eyes for subconjunctival hemorrhage, which may be a sign of coagulopathy or retrobulbar hemorrhage.
- Ask the patient to open the mouth and put out the tongue. Look for signs of angioedema or any oral bleeding.
- Inspect the neck for raised jugular venous pressure (JVP)/distended veins, which may indicate either right ventricular (RV) failure or superior vena cava syndrome. Palpate the anterior neck for signs of subcutaneous emphysema or any swelling that may obstruct the airway.
- Examine the limbs and skin for new-onset rash, signs of cellulitis or necrotizing fasciitis, compartment syndrome, limb ischemia, or septic arthritis. Special care should be taken to examine the pressure points for ulcers, which should include turning the patient carefully to examine the back and sacrococcygeal area.
- Examine the genitalia for signs of orificial bleeding, infection, and obstructed or strangulated hernia.
- The physical examination should conclude by taking note of the invasive lines/devices that are present, their site of insertion, day of insertion, and need for further presence.

Systemic Examination

Neurological examination: Examining a critically ill nonintubated patient is similar to any other outpatient with the caveat that many of the physical signs may not be elicited due to noncooperation and frailty of the patient.

- GCS/FOUR score should be calculated at the baseline and at regular intervals.
- Confusion Assessment Method for the ICU (CAM-ICU) or the Intensive Care Delirium Screening Checklist (ICDSC) should be used to confirm the presence of delirium.
- Patient should be examined for orientation to time, place, and person. Ask the patient to perform few simple movements or answer simple questions.
- Assess for the presence of pain using validated tools such as 0–10 Numeric Rating Scale (NRS), Behavioral Pain Scale-Non Intubated (BPS-NI) and the Critical Care Pain Observation Tool (CPOT). All communication should be done in a language that the patient understands.
- Examine the eyes for nystagmus and ophthalmoplegia that may be signs of stroke, meningoencephalitis, or raised ICP due to any cause.
- Pupils need to be examined for their size, shape, reactivity, and symmetry on both sides. Miotic pupils may indicate opiate overdose, organophosphate poisoning, or pontine stroke among other various causes. Dilated pupils may be seen with anticholinergic

drug toxicity, metabolic encephalopathy, neurotoxic snake bite, etc. Asymmetric nonreactive pupils are an ominous sign that indicates uncal herniation, which needs urgent brain imaging and intervention.

- Look for lower cranial nerve palsies, which may indicate brainstem stroke by asking the patient to say "ahh" and observing the movement of soft palate, asking the patient to protrude the tongue out, shrugging of shoulders, assessing the ability to swallow liquids, and to cough out secretions.
- A bedside fundoscopy can be performed to look for papilledema secondary to raised intracranial pressure (ICP) and subhyaloid retinal hemorrhage due to subarachnoid hemorrhage.
- Assess the symmetry and power of movements of all four limbs including the strength of hand grip.

Other Systems

- Auscultation of the chest is required to listen for wheeze which could be bilateral/unilateral and monophonic/polyphonic. The presence of wheeze usually indicates bronchospasm/fixed obstruction of airways. Also listen for crepitations indicating pulmonary edema (cardiogenic/noncardiogenic), bronchial breath sounds (consolidation), and absent breath sounds (pneumothorax, life-threatening asthma, or pleural effusion).
- Cardiac auscultation needs to be done to listen for new-onset murmurs (MI or infective endocarditis) and muffled heart sounds (pericardial effusion/tamponade).
- Inspect and palpate the abdomen to look for distension, discoloration, presence of stoma or drainage tubes, tenderness, guarding, or rigidity. Acute encephalopathy may be precipitated (especially in the elderly) due to acute abdominal conditions such as cholecystitis, pancreatitis, perforated viscus, bowel ischemia, paralytic ileus, intestinal obstruction, intra-abdominal infections, and complicated urinary tract infection (UTI).

Adjuncts to Bedside Examination

Point-of-care ultrasound (POCUS) and arterial blood gas (ABG) analysis are an integral part of routine bedside assessment. ABG provides information regarding hypoxemia, hypercapnia, metabolic acidosis/alkalosis, hyponatremia, hypernatremia, hypercalcemia, anemia, glucose, and lactate levels. These parameters can aid in arriving at an etiology for the altered mental status.

Point-of-care ultrasound aims at bedside identification/assessment of important factors that may lead to life-threatening emergencies. In case of acute encephalopathy associated with hypoxia, shock or both, bedside echocardiography, and lung and vascular ultrasound are essential adjuncts. Echocardiography should be done to look for LV contractility (commonly by eyeballing method), presence of intracardiac thrombus/vegetations, pericardial effusion/tamponade, RV dilatation (pulmonary embolism), and ascending aortic dissection involving the aortic root. Lung and vascular ultrasound should be done to look for presence of pneumothorax, pulmonary edema, consolidation, hemothorax, and deep vein thrombosis.

Relevant Investigations

The choice of investigations depends on the history and physical examination findings along with the results of POCUS and ABG analysis. It needs to be individualized.

- Complete blood count—anemia/polycythemia, leukocytosis/leukopenia (infections), and thrombocytopenia.
- Metabolic panel [blood urea nitrogen (BUN), serum creatinine, sodium, potassium, chloride, magnesium, calcium, liver function tests, serum ammonia, and serum osmolality].
- Coagulation screen in case of suspected coagulopathy or intracranial bleeding [prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (APTT), D-dimer, and fibrinogen].
- A 12-lead ECG and serum troponins [if myocardial infarction (MI) or acute heart failure is suspected].
- Blood, bronchoalveolar lavage (BAL), and urine culture in case of suspected infection at those sites.
- Lumbar puncture and cerebrospinal fluid (CSF) analysis for suspected meningoencephalitis.
- Computed tomography (CT)/magnetic resonance imaging (MRI) of brain for acute stroke (hemorrhagic/ischemic), hypertensive encephalopathy, posterior reversible encephalopathy syndrome (PRES), intracranial space-occupying lesion, and subarachnoid hemorrhage.
- Serum/urine toxicological screening as per the differential diagnoses (ethanol, methanol, ethylene glycol, acetaminophen, salicylate, and valproate).
- Ultrasonography (USG)/CT abdomen in case of suspected intra-abdominal pathology.

Diagnosis of Delirium

Delirium screening needs to be performed daily for every patient admitted in the intensive care unit. Screening should be done using validated tools such as CAM-ICU or the ICDSC. These tools are based on assessment of attention and cognition and fulfil certain points of the aforementioned DSM-5 criteria if positive. In addition, it is important to establish that the acute encephalopathy is not due to prior neurocognitive deficit and that the patient is not comatose. It is also necessary that the acute encephalopathy is secondary to a precipitating factor. Any set of symptoms that are suggestive of delirium but do not fulfil the criteria should be termed as "subsyndromal delirium".

CASE SCENARIO 2

A 45-year-old female was admitted to the surgical ICU after undergoing Whipple's procedure for pancreatic cancer. She was mechanically ventilated and was requiring vasopressor support. She was receiving fentanyl with midazolam for analgosedation. On postoperative day 3, sedation was stopped, and a spontaneous breathing trial was given. However, the patient kept biting on the tube and was visibly agitated.

Screening

The approach to "acute encephalopathy" or delirium in a patient who is intubated and has been in the ICU for quite some time is very different from that in a conscious nonintubated patient. In this scenario, the first step is to diagnose/detect the presence of delirium and then proceed toward finding an etiology for the same. This can be achieved only if all the intubated patients are screened every day for the development of delirium. Screening is done by either the CAM-ICU or ICDSC method. Daily awakening by sedation hold is one of the key aspects that facilitate weaning and prevents complications like ventilator-associated pneumonia (VAP). It is during this awakening trial that the patient needs to be screened for delirium. Once it has been established that a patient has delirium or at least subsyndromal delirium, the next step is to find the factors contributing toward it. The etiology is usually multifactorial, and each factor needs to be addressed separately. Even in the absence of delirium, a diagnosis of acute encephalopathy is important as many factors can lead to an agitated intubated patient.

Focused Assessment

The assessment can be carried out as per the broad outline given below:

- The vitals signs on the monitor need to be noted for hypoxia (SpO_2), hypotension, hypertension, tachyarrhythmias, bradyarrhythmias, end-tidal CO_2, and fever (if a temperature probe is attached).
- Observe the patient for a minute and take note of all the invasive lines or devices. All foreign devices such as central venous catheters, peripheral venous catheters, arterial catheters, urinary catheter, endotracheal or tracheostomy tube, intercostal chest tubes, and abdominal drains are potential sources of infection, which is an important risk factor for delirium. Note the duration for which the line/tube has been in situ and the need for its further presence.
- One needs to pay attention to the ventilator and its circuit. Any obstruction in the endotracheal/tracheostomy tube and the circuit due to secretions, blood, water condensate, or kinking can lead to agitation in the patient as it increases the resistance and work of breathing. Review the mode of ventilator, settings, and the graphs/loops on the ventilator screen. Identification of various types of ventilator asynchronies is essential for its early correction and prevention of ventilator-induced lung injury as well as agitation/delirium.
- Assessment of pain is an important but often neglected aspect of care of an intubated patient. The assessment should be done by validated tools such as Behavioral Pain Scale (BPS) or CPOT. An appropriate analgesic or multimodal analgesia and the required doses need to be administered followed by frequent reassessments to ensure adequacy of pain relief.
- The ABG analysis provides valuable information regarding the acid–base disturbance, adequacy of ventilation, oxygenation status, anemia, electrolyte imbalance, and hypoperfusion. Correlate the ABG with the ventilator settings to make necessary adjustments as respiratory acidosis and hypoxemia are common causes for agitation and increased respiratory drive. Both metabolic acidosis and alkalosis need to be investigated and corrected if present.
- An often underrecognized cause of delirium or agitation in an intubated patient is persistent thirst and/or hunger. Assessing thirst or hunger is difficult because of the inability to communicate. Presence of hypernatremia in the absence of external sodium loading is usually a sign of free water deficit. This free

water deficit is a strong stimulus for thirst and remains persistent if left uncorrected. Hence, hypernatremia should always be aggressively corrected to prevent persistent agitation or delirium. Similarly, hunger assessment does not have a readily available external marker. Hence, the standard practice of starting enteral nutrition as early as possible with adequate calories should be practiced at all time along with monitoring of blood glucose.

- Certain seemingly minor things that actually play an important role in patient comfort are patient position in the bed, bowel movements, fever, minimization of ambient lights and noise at night, communication in a familiar language, provision of hearing/visual aids that are used by the patient, and provision of means to communicate like a pen and paper.
- After the above points are considered and corrected, the remaining head-to-toe assessment is like that in a nonintubated patient as discussed previously. The goal of this head-to-toe examination is to find a source of infection, inflammation, or trauma that may precipitate or contribute to the ongoing agitation or delirium.
- Once all the probable causes are ruled out, many patients have an increased respiratory drive secondary to their underlying severe critical illness. Such patients need adequate sedation to ensure patient–ventilator synchrony. Hence, sedation should always be used as a measure of last resort and not as a knee-jerk reflex to an agitated patient!

Chart Review

The bedside charts of the patient need to be reviewed to get an idea of the trend of vital parameters and/or blood investigations, hourly fluid intake/output, and the frequency/consistency of bowel movements throughout the ICU stay. The importance of reviewing the current medications, their doses, and the need for further use cannot be overemphasized.

SUMMARY

Detection of acute encephalopathy in an intubated patient is the first step, which should then trigger a search for the causes. Delirium screening should be done as per the CAM-ICU or ICDSC method and a formal diagnosis of delirium made only if the patient meets the DSM-5 criteria. The etiology is often multifactorial, and all the factors need to be managed to collectively treat delirium and its associated complications.

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Chapter

33

How will I Approach/Examine a Patient with New-Onset Fever?

Thomas Sudarsan, Lovely Thomas

ABSTRACT

New-onset fever in an intensive care unit (ICU) patient requires careful consideration of history, review of records, and a complete physical examination followed by judicious use of investigations to arrive at a diagnosis. Considering the significant adverse consequences of infectious causes secondary to hospital-acquired infections, the focus needs to be directed primarily towards these diagnoses while at the same time considering noninfectious causes also. The use of scores and ancillary skills such as point-of-care ultrasound add value and confidence in empirical management based on clinical judgment.

INTRODUCTION

New-onset fever in an intensive care unit (ICU) denotes fever in a patient after 48 hours of an afebrile course in ICU **(Fig. 1)**. The American College of Critical Care Medicine (ACCCM) and Infectious Disease Society of America (IDSA), joint task force, defines temperatures above 38.3°C or ≥101°F as a fever that needs further evaluation. A temperature >100.4°F (38°C) may be significant in immunocompromised patients, elderly patients, or who are on acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs).

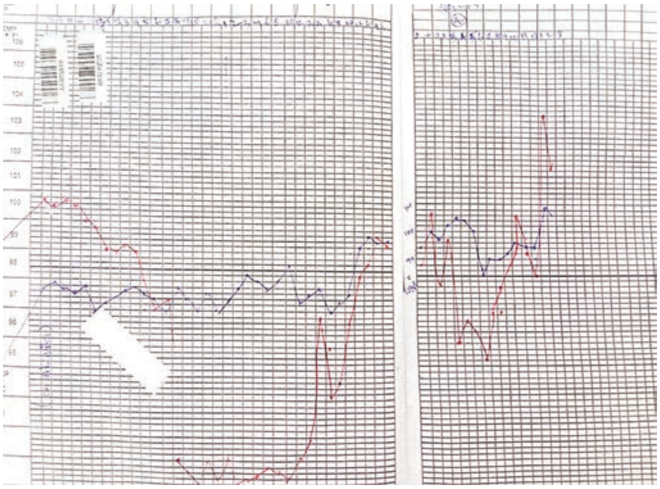


Fig. 1: New-onset fever in intensive care unit.

Measurement of core temperatures is preferred. Consistency in the method adopted will help in following the trend of fever. Rigors represent the hypothalamic response to an increase in body temperature and often precede the development of a fever. Rigors are related to bacteremia and a patient with new-onset rigors should be investigated.

A temperature of >105.8°F (or 41°C) is called hyperpyrexia or hyperthermia. Hyperthermia is caused by increased temperature generation or decreased heat dissipation as opposed to fever due to an altered hypothalamic set point. Hyperthermia has a worse prognosis and needs urgent temperature control in all patients.

Hypothermia is generally accepted to mean a core body temperature <35°C. Hypothermia is encountered less frequently than fever in critically ill patients and may reflect a myriad of clinical conditions. Detailed discussion on hypothermia is beyond the scope of this chapter.

CASE SCENARIO

A 65-year-old male with diabetes had presented with altered sensorium and was intubated because of respiratory failure and severe high anion gap metabolic acidosis. He was found to have Staphylococcus aureus bacteremia. He had a history of fever with upper respiratory symptoms before admission for which he had taken treatment from a physician with oral azithromycin for 5 days. He was intubated, started on high-dose intravenous cloxacillin, and stabilized with *supportive care. After a period of defervescence of 7 days, he developed a new-onset fever and shock.*

On examination, he had a temperature of 102°F, heart rate was 62 beats/min, systolic blood pressure (BP) of 90/40 mm Hg, and respiratory rate of 30 breaths/min. Skin and soft tissue examination revealed no rash, all peripheral joints were normal, and no bed sore. Respiratory examination revealed equal air entry with no added sounds, his ventilatory requirement was stable on 30% FiO_2 and positive end-expiratory pressure (PEEP) 6 cmH_2O. Cardiovascular examination showed warm peripheries with a new early diastolic murmur in the left third intercostal space. Abdomen and central nervous system (CNS) examination were normal.

How would You Approach this Patient?

History and Symptomatology

A thorough review of clinical history starting from when the patient was of good health, presenting illness in chronological progression, and history of other chronic medical conditions is imperative and helps to have an algorithmic approach and hence avoid unwarranted investigations in the pursuit of a diagnosis. History taking should also include detailed drug history, history of previous drug reactions, and any addictions. Retaking and reviewing history (at times from multiple, relevant caretakers) will be crucial in differentiating a new-onset fever versus relapsing pyrexia of unknown origin.

A systemic approach is essential while evaluating the causes of the new onset of fever. A structured and comprehensive anatomical approach to examination helps narrow down the differentials for infective or noninfective causes of fever.

The examiner should follow professional etiquette by introducing himself, obtaining consent, and ensuring the privacy and comfort of the patient in the presence of a chaperone, especially during sensitive examinations. The clinical examination starts with appropriate exposure of the patient for a thorough examination and subsequent protection to prevent hypothermia and maintain the dignity of the patient.

Physical Examination

Initial approach: As in any critically ill patient, airway, breathing, and circulation takes importance and patient resuscitation should be done while staying focused on the possible causes and eliminating them by thorough clinical examination and appropriate investigations. If a septic shock is suspected, representative cultures should be taken and antibiotics started.

General examination: The general examination will give ample clues regarding the source of the infection. The examination does not stop with head-to-toe screening, but rather should include examination of lines and tubes, patient environment, and daily progress charts. Moreover, the examination should be the systematic and focused as described in **Table 1**.

Systemic Examination

Respiratory system: Infection-related ventilator-associated condition (IVAC) is considered in presence of <36°C or >38°C temperature or elevated leukocyte counts *and* ≥1 antibiotics continued for ≥4 days within 2 days before or after VAC onset excluding the first 2 days of mechanical ventilation. Ventilator-associated condition (VAC) is respiratory derangement in a patient who had a minimum of 2 days of mechanical ventilation with stable or decreasing oxygen requirements before the days of increased oxygenation defined by an increase in FiO_2 by 20% or rise in PEEP by 3 cmH_2O for ≥2 days. An increase in the volume or change in the consistency of endotracheal secretions, new-onset infiltrate, or opacity on a chest X-ray (CXR; **Fig. 2)** will also indicate a probable IVAC. In scenarios fulfilling the conditions for IVAC, an endotracheal aspirate sample is warranted for Gram stain and culture before starting antibiotics.

Patients admitted with community-acquired pneumonia on an appropriate antibiotic, developing change in intensity or pattern of fever, should be screened for complicated pleural effusion/empyema or lung abscess in which case diagnostic pleural tap and a computed tomography (CT) thorax imaging may be warranted.

Noninfective cause of fever secondary to pulmonary causes such as acute respiratory distress syndrome and cryptogenic organizing pneumonia needs to be considered if pulmonary secretions culture persistently come negative and radiological images consistent with the same.

Cardiovascular system: A careful review of monitoring sheets can likely unveil variation in trend of vitals, such as heart rate, pulse pressure, and increasing fluid balance with edema, which might give early clues to the development of a new onset infection. Change in intensity of heart sounds or new onset murmur or finding of elevated jugular venous impulse in a patient with

TABLE 1: Sequential head-to-toe examination of a patient with new-onset ICU fever.

Examination of the patient in the supine position		
Head and neck	- *Eye and fundus:* Petechiae and Roth's spots, endophthalmitis, CMV retinitis - *Ear, sinus, and mastoid:* Tenderness, mucopurulent/blood-stained discharge—sinusitis (bacterial/fungal), acute otitis media, sialadenitis, CSF rhinorrhea predisposing to nosocomial meningitis - *Neck stiffness:* Meningitis/subarachnoid hemorrhage	
Torso	- *Thorax:* Petechia, herpetic rash, intercostal tenderness—empyema/liver abscess - *Cullen and Grey Turner sign:* Hemorrhagic pancreatitis - *Generalized:* Abdomen tenderness, guarding and rebound tenderness—peritonitis or mesenteric ischemia - *Focal: renal angle:* Pyelonephritis, right hypochondriac—cholecystitis, suprapubic fullness, and tenderness: CAUTI/diverticulitis	
Limbs	- *Limb girth asymmetry:* DVT supplemented by compression sonography of lower limb veins - Large peripheral joints need to be inspected for effusions with patellar tap or painful restrictions of joint movements to consider metastatic septic or reactive arthritis	
Genitalia	- Skin necrosis with underlying dead tissue—Fournier gangrene - *Vesicular lesions:* Reactivation of herpes, purpura fulminans - *Scrotal swelling with testicular tenderness:* Epididymo-orchitis, polyarteritis nodosa, pyocele - *Foul-smelling vaginal secretions in postpartum patients:* Differentials—septic abortion or endometritis - Look for pus discharge from the urethral meatus—urinary tract infection	
Lines and tubes: Daily review of need and consideration of removal if unnecessary		
Tubes	ET	*ET:* Secretions, change in quality and quantity, frequency of suctioning associated with other markers of VAP
	Nasogastric tube	*Sinus tenderness:* Ultrasound of the sinus can reveal mucosal thickening or air-fluid level
	Urinary catheter	- Suspect UTI in obstructed system. Note also for change in color or hematuria - Infective or noninfective (Cyclophosphamide induced cystitis, immune glomerulonephritis)
Lines	CVC	Note the day of the vascular access and always question the need and consider removal
	Dialysis port	Lines inserted in any emergency setting should be considered for reinsertion/removal under aseptic conditions within 24 hours once the patient is considered stable
	Peripheral venous cannula	Inspect transparent dressing for signs of inflammation or extravasation of fluids/medications although these findings are not consistent
	Arterial cannula	For arterial cannula, look at the site of entry and for signs of vascular compromise, i.e., digital cyanosis and gangrene
Skin	Generalized rash	Manifestation of the primary cause of fever like rickettsial infection/varicella/staphylococcal scalded skin syndrome/meningococcal rash
	Localized skin redness and bullae with undue tenderness	*Secondary:* - Drug rash/purpura fulminans - If differential include vasculitis, SJS/TEN, consider a biopsy and dermatology opinion
	Postoperative surgical wound	- Consider cellulitis/necrotizing fasciitis - Daily inspection of the surgical wound and drain content helps to avoid missing surgical site Infection or leak of anastomosis
Examination of the back		
Posterior and perineal region	- *Pressure sore:* Watch for progression of inflammation, adequacy of granulation, and change in color - *Spine tenderness with Gibbus:* Infective spondylodiscitis - Renal angle tenderness for pyelonephritis - Inspect perianal region and perform per rectal exam (PR) for fistula in ano and ischiorectal abscess (Caution with PR in neutropenic patients)	

(CAUTI: catheter-associated urinary tract infection; CMV: cytomegalovirus; CSF: cerebrospinal fluid; CVC: central venous catheter; DVT: deep vein thrombosis; ET: endotracheal tube; ICU: intensive care unit; SJS: Stevens–Johnson syndrome; TEN: toxic epidermal necrolysis; UTI: urinary tract infection; VAP: ventilator-associated pneumonia) heart failure should alert us to the possibility of infective endocarditis as in our patient with staphylococcal bacteremia or candidemia. In such a patient peripheral finding of jaundice, splinter hemorrhages, Janeway lesions (Nontender irregular hemorrhagic macules in palms and soles representing septic microemboli), Osler's nodes (Tender appendicular nodules representing vascuilitic immune phenomenon), and septic embolic infarcts with tender hepatosplenomegaly with a rub on auscultation will help to fortify the clinical diagnosis. Transthoracic echocardiogram (ECHO) or a transesophageal ECHO will help confirm the diagnosis **(Figs. 3A and B)**. An electrocardiogram (ECG) showing a new-onset bradycardia with PR interval prolongation may alert to an aortic root abscess in the appropriate clinical setting.

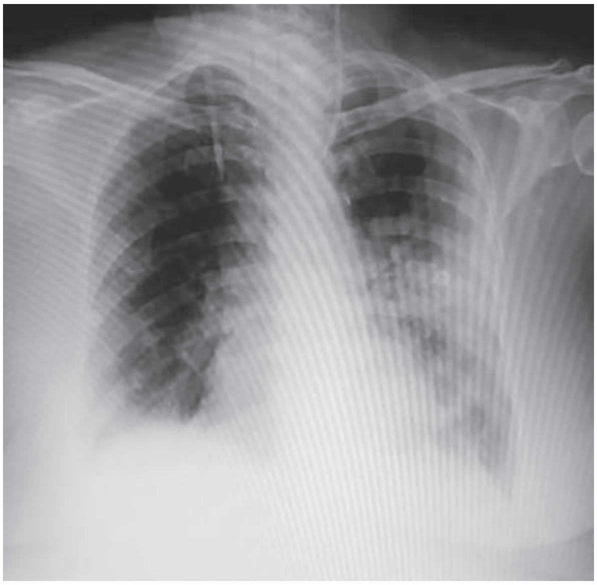
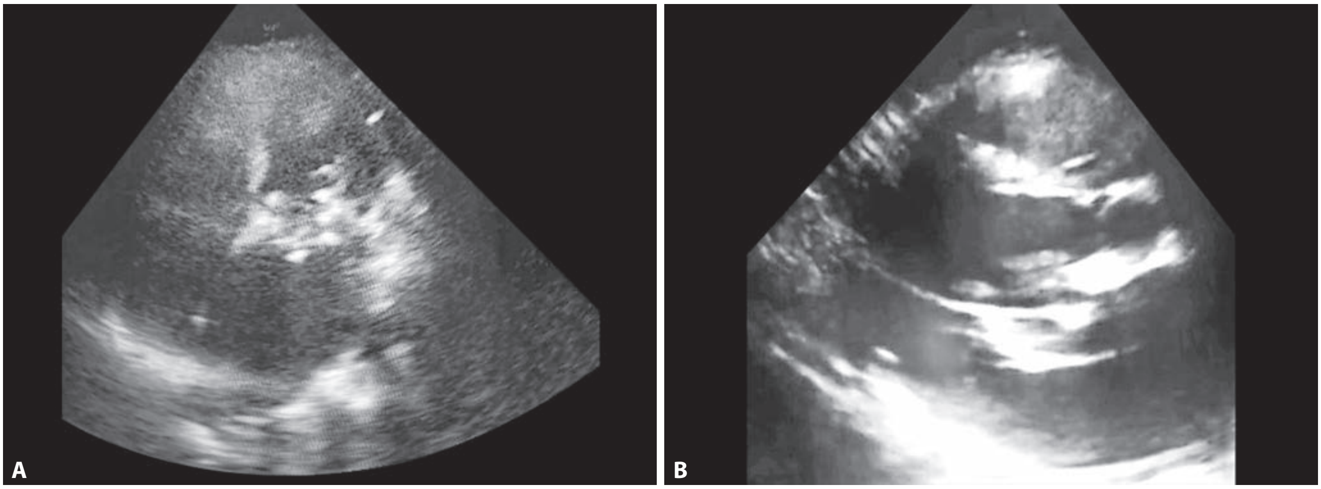


Fig. 2: Left lower lobe consolidation in a patient with ventilator-associated pneumonia (VAP).

Gastrointestinal system: Bedside examination includes scrutiny for scleral and cutaneous jaundice, abdominal distention, palpation for tenderness of solid organs, as well as auscultation for the presence of bowel sounds and rubs. The emergence of new-onset ileus and tenderness on abdomen palpation with or without the onset of jaundice should make us vigilant about the abdomen as the root cause of fever, which might be infective or sterile. Pancreatitis, acalculous cholecystitis, metastatic liver abscess, mesenteric ischemia, perianal abscess, or an infected bedsore are a few differentials to consider and to look for in imaging studies.

A per-rectal examination in a male or a per-vaginal examination in a female might reveal a tender prostatic abscess or a pelvic hematoma/collection although such an examination should be done after due consideration in a neutropenic patient for fear of bloodstream translocation. Patients on broad-spectrum antibiotics with new onset watery stools mixed with pus or blood should be isolated and promptly evaluated for probable *Clostridium difficile* colitis while evaluating for other causes noninfective. There is clearly no value in requesting a stool culture for evaluation of fever in an ICU except in an immunocompromised patient and in the setting of an institutional outbreak of gastroenteritis.



Figs. 3A and B: Infective endocarditis with vegetation in a prosthetic mitral valve.

Neurological system: Daily spontaneous awakening trial if safe and logistically possible can help evaluate and document any change or trend of neurological status on a scheduled basis. Though a detailed neurological examination may be out of scope in a sedated and ventilated patient, cardinal examination with the Glasgow Coma Scale, pupillary size and reaction to light, asymmetry/abnormal tone, differential power and reflex and extensor plantar response, will shed light on any neurological (central and peripheral nervous system) involvement. For example, our patient with staphylococcal bacteremia due to pneumonia can develop metastatic complications of the infection with spondylodiscitis or mycotic aneurysm with cerebrovascular accident, which can present as weakness of a limb or hemiparesis. Such complications have been described in various suppurative conditions such as infected pancreatic collections, empyema, or emphysematous pyelonephritis.

A reset of the hypothalamus target temperature to a higher level is responsible for fever in central/neurogenic etiology. It is a diagnosis of *exclusion*. Patients at risk include patients with traumatic brain injury with diffuse axonal injury or frontal injury, subarachnoid hemorrhage, intracerebral hemorrhage, acute ischemic stroke, brain tumor, or patients with surgery or pathology involving the hypothalamus. Fever has its onset within 72 hours of admission with sustained, continuous spikes without diurnal variations and relative bradycardia (Every degree rise in Fahrenheit temperature accompanied by pulse increases of 10 beats/min being the norm in fever). There is a notable absence of perspiration and poor or no response to antipyretics. The lack of definitive diagnostic criteria makes it even more challenging in recognizing the condition. A high index of suspicion in a clinical setting known to be associated with the diagnosis coupled with careful clinical assessment will help clinch the diagnosis.

Drug chart: Daily inquisitive survey of the drug chart and elicitation of complete drug history helps to avoid missing drug fever or the possibility of severe idiosyncratic reactions. Drug fever is also a diagnosis of exclusion. Its onset can be varying from a few hours to months following initiation of the offending agent and can potentially happen with the intake of chronic drugs. Fever is characterized by fluctuating fever with wide swings that do not return to baseline temperature and may accompany a rash. They occur more frequently in certain patient populations, such as people with human immunodeficiency virus (HIV), underlying malignancy, and liver or renal transplants. The white blood cell count can be elevated with eosinophilia. Resolution of drug fever will occur within 72–96 hours of discontinuing the offending drug. Fever with confusion, muscle rigidity, and/or dysautonomia should raise suspicion for a severe idiosyncratic reaction. It has been commonly linked to anticonvulsants such as carbamazepine, phenytoin, phenobarbital, and antibiotics such as beta-lactams, sulfonamides, and nitrofurantoin.

Other syndromic presentations of neuroleptic malignant syndrome, sympathomimetic, anticholinergic, and serotonin syndrome should be considered in patients presenting with suspected drug overdose and hyperthermia. **Table 2** summarizes the infectious and noninfectious causes of new-onset fever. This list is by no means exhaustive but just provides a brief overview of the common differentials to consider.

What are the Possible Differentials to be Considered?

On review of the history, patient had presented with probable viral pneumonia with secondary bacterial staphylococcal infection and diabetic ketoacidosis being precipitated by the infection. He subsequently developed a new-onset fever after a period of quiescence. The differentials to consider here are:

- *Infective cause:*
  - New infection, which is hospital-acquired infection, such as ventilator-associated pneumonia (VAP), catheter-related bloodstream infection (CRBSI), and catheter-associated urinary tract infection (CAUTI)
  - Inappropriate treatment of primary infection—inadequate dosing, resistant *Staphylococcus*
  - Sequestered collection, suppurative complication, and infective endocarditis
- *Noninfective cause:*
  - *An immunological complication of the preceding infection:* Reactive arthritis, thrombotic microangiopathy
  - *Drug fever:* Penicillin
  - Deep vein thrombosis (DVT) with pulmonary embolism contributing to shock.

Investigations should be tailored based on the clinical findings and the differentials considered.

Our patient's blood culture was later speciated to methicillin-susceptible Staphylococcus aureus (MSSA) with a low minimum inhibitory concentration (MIC) ≤0.5 µg/mL

TABLE 2: List of infective and noninfective causes of new-onset fever in ICU.

	Infectious causes	***Noninfectious causes***
Respiratory	• Ventilator-associated tracheobronchitis • Ventilator-associated pneumonia • Complicated syn-pneumonic infection	• Cryptogenic organizing pneumonia • ARDS • Aspiration pneumonitis
Abdomen	• Acalculous cholecystitis • Liver abscess • *Clostridium difficile* colitis	Pancreatitis
Cardiovascular	Infective endocarditis	• Pulmonary embolism • Deep venous thrombosis **(Fig. 5)**
Tubes and lines	• Catheter-related bloodstream infections • Catheter-associated urinary tract infection	Thrombophlebitis **(Fig. 6)**
Skin and Joints	• Bed sore **(Fig. 4)** • Purpura fulminans • Cellulitis or necrotizing fasciitis • Surgical site infection • Septic arthritis	Drug rash with penicillin, phenytoin, etc.
Central nervous system	• Metastatic seeding of infection—meninges/spinal • Brain abscess	Neurogenic fever
Endocrine		• Thyrotoxicosis • Pheochromocytoma • Adrenal crisis
Drug related		• Drug fever • Malignant hyperthermia • Neuroleptic malignant syndrome • Serotonin syndrome
Procedure related		• Febrile transfusion reaction (within 6 hours of transfusion) • Benign postoperative fever

(ARDS: acute respiratory distress syndrome; ICU: intensive care unit)

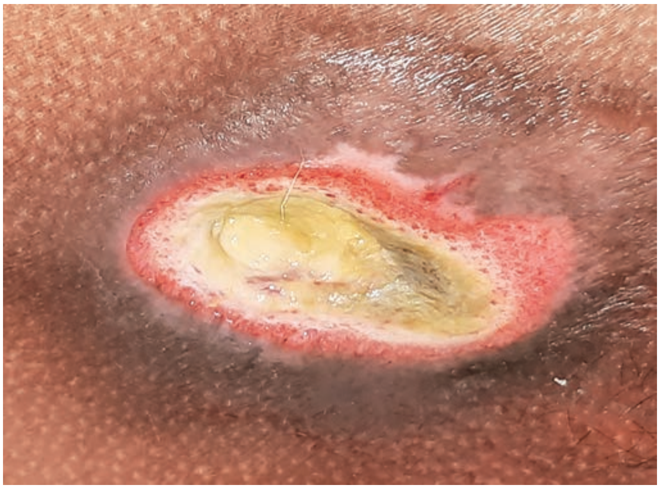


Fig. 4: Infected decubitus ulcer.

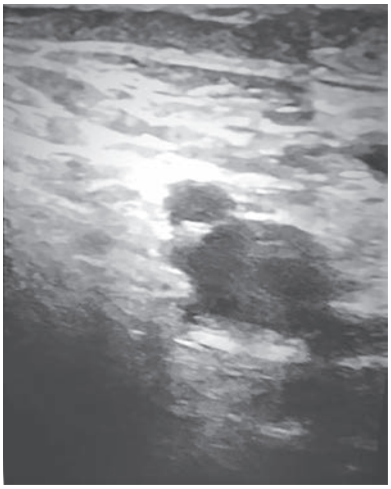


Fig. 5: Noncompressible or visualized thrombus in femoral vein suggestive of deep vein thrombosis.

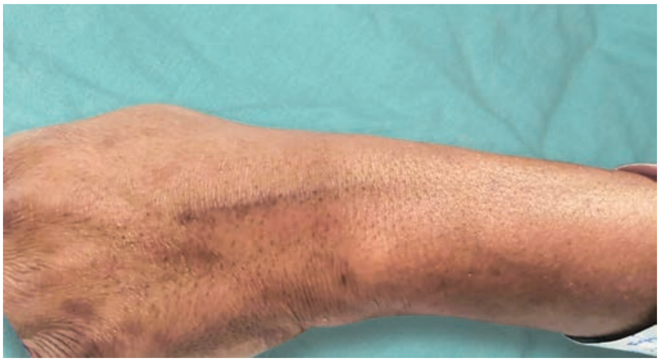


Fig. 6: Infective thrombophlebitis at vascath site.

for oxacillin, hence he was on appropriate antibiotics. The repeat cultures sent after 72 hours of treatment showed clearance. There was no evidence to suggest IVAC or CRBSI or CAUTI. Given fever with shock, paired cultures were taken, central venous catheter (CVC) was resited, and he was empirically added on polymyxin and vancomycin, awaiting cultures.

ECG taken because of shock showed PR interval prolongation. ECHO showed a bicuspid aortic valve with a suspicious aortic root abscess. An urgent cardiothoracic consult was sought for.

SUMMARY

Evaluation of the new onset of fever in the ICU requires a diligent general and systemic examination to refine and narrow down the clinical diagnosis based on which further confirmatory/supplementary investigations will be directed. Along with infective, noninfective causes also should be considered to avoid unnecessary laboratory investigations and antibiotic misuse which might lead to adverse patient outcomes.

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Chapter

34

How will I Approach/ Examine a Patient with Refractory Hypoxemia?

Sumalatha Arunachala, Jeevan Jagadeesh

ABSTRACT

This comprehensive review focuses on the clinical assessment and management of refractory hypoxia in critically-ill patients. The chapter highlights the importance of a detailed patient history in identifying the underlying causes of hypoxemia. Clinical examination is a fundamental component of the assessment process. The SAVE (scalar assessment of ventilator during emergencies) protocol is introduced as a valuable tool for optimizing ventilator settings in patients with refractory hypoxemia. The use of electrical impedance tomography (EIT) when available to detect shunt and dead space pathologies is explained. Monitoring peak and plateau pressures is emphasized, and the use of esophageal pressure monitoring is discussed. Lung ultrasound offers a noninvasive, real-time assessment of lung aeration and pathology, aiding in the identification of underlying causes contributing to refractory hypoxemia along with evaluating the efficacy of therapeutic interventions, such as positive end-expiratory pressure (PEEP) adjustment or recruitment maneuvers. Interpretation of arterial blood gases is crucial for assessing the patient's respiratory status and guiding management decisions. The integration of history, bedside clinical assessment, and use of adjuncts such as EIT and echocardiography, assessment of ventilator scalars, interpretation of arterial blood gases, and utilization of lung ultrasound plays a crucial role in diagnosing the underlying cause, optimizing treatment, and improving patient outcomes. Esophageal pressure monitoring has an important role in the ventilatory management of acute respiratory distress syndrome (ARDS) patients.

INTRODUCTION

Hypoxia, refractory to conventional therapies, poses an immediate threat to end organs and unless tackled within minutes, can lead to devastating loss of life. Clinician's knowledge, skills, and presence of mind are pivotal to a patient's survival. An in-depth understanding of the causes of refractory hypoxemia, a short but well-directed history-taking and clinical examination, and the use of appropriate gadgets available at the bedside [arterial blood gases analysis, ultrasonography, electrical impedance tomography (EIT), and esophageal pressure monitoring] will give enough clues to the underlying cause for hypoxemia. In this chapter, we will focus on how to examine a patient with refractory hypoxia and discuss on how to approach a patient having refractory hypoxemia leading to bedside clinical diagnosis.

CASE SCENARIO

Mrs N, a 38-year-old female, who is obese, has a known case of bronchial asthma (BA), and rheumatoid arthritis on oral steroids was admitted to the intensive care unit (ICU) with 5 days of fever, cough, and 2 days of breathlessness. The mother informed you that she has frequent hospital admissions for BA. She received treatment from the primary health center treated with third generation cephalosporin, doxycycline, paracetamol, oxygen, and intravenous (IV) fluids. Given worsening breathlessness, she was referred to your hospital. In the emergency department, she is intubated given impending respiratory failure and shifted to ICU. Currently, she is intubated, sedated, and paralyzed. Her oxygen support includes fraction of inspired oxygen (FiO_2) *at 100% and positive end-expiratory pressure (PEEP) at 12 cm*H_2O*. After 8 hours of ICU treatment, her oxygen saturation* (SpO_2) *is 93%, and the arterial partial pressure of oxygen* (PaO_2)/FiO_2 *(P/F) ratio is 90.*

WHAT DO YOU DO NEXT?

Although the history is grossly inadequate at this point, it is important to treat the life-threatening severe hypoxemia. Refractory hypoxia is a clinical condition considered when the oxygen levels are very low for a longer time despite ventilator support and high oxygen delivery. There are no definitive definitions and most of the time we adapt from acute respiratory distress syndrome (ARDS) literature. We can consider it as refractory when the SpO_2 is <88–90% or PaO_2 <55–60 mm Hg despite optimal treatment.

Refractory hypoxia can be considered when:

- Persistent hypoxia despite correction or exclusion of common reversible causes, e.g., pneumothorax, equipment failures.
- PaO_2/FiO_2 ratio < 100 mm Hg despite optimal ventilator settings, i.e., FiO_2 > 0.6 (60% oxygen) and PEEP >10 cmH_2O.
- If hypoxemia persists for >6–8 hours despite appropriate evaluation and intervention.
- Minimal or no improvement with conventional strategies (e.g., recruitment maneuvers, prone ventilation).

It is important to note that the exact definition can vary slightly between institutions and studies. The key concept is the persistence of hypoxemia despite appropriate interventions, indicating a severe underlying pathology or complex physiological derangement. In this chapter, we consider refractory hypoxia if any of the above criteria are met.

When examining such patients, the main goals should be to determine the reason for the refractory hypoxia, look for additional mechanisms, look for indicators of systemic disorders that could affect the oxygenation, and keep an eye on the current course of therapy. *See* **Table 1** for a summary of initial patient assessment and emergent management.

TABLE 1: Emergent and urgent steps to ensure adequate oxygenation in Mrs N.

Emergent measures	***Explanation***
Ensure the appropriate tracheal position of the endotracheal tube	It is important to rule out endobronchial or esophageal intubation, tube migration, or extubation during the transport of the patient
Ensure the patency of the endotracheal tube by performing suctioning	Mucous plugging/blood clot can obstruct ventilation
Disconnect from the ventilator and connect with AMBU bag with PEEP valve and ventilate with 15 L of oxygen	Removes all ventilator-related causes of hypoxemia
Check the integrity of the endotracheal cuff	Rules out the faulty endotracheal tube with cuff malfunction/cuff tear during intubation (sharp teeth of patient may sometimes damage the cuff)
Check hemodynamics	Significant bradycardia or hypotension can cause low saturation
Check the waveform on the pulse oximeter	Poor perfusion due to cold peripheries can lead to the inappropriately low reading of the percentage of saturation
Ensure adequate handover from emergency resident	• What was Mrs N's saturation when she came to the emergency? • What were the drugs used for endotracheal intubation? • How was the tracheal intubation confirmed? • Was it a difficult airway? • Was there any bleeding from the endotracheal tube? • How were the secretions? Thick? Bloody? • Was there any evidence of aspiration? • Was there any tooth missing? • At what level was the endotracheal tube fixed? • How was the hemodynamics? • Was there any trouble during transport?
Urgent measures	***Explanation***
Follow BLUE protocol	Leads to spot bedside diagnosis of the cause of hypoxemia such as pneumonia, ARDS, asthma/COPD, pneumothorax, pulmonary edema, and pulmonary embolism
Perform arterial blood gas analysis	Essential to calculate P/F ratio, oxygenation index, $PaCO_2$-$EtCO_2$ gap, and A-a gradient

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Urgent measures	***Explanation***
Perform screening echocardiography	Recognizes intracardiac major shunts, corroborates acute pulmonary embolism, detects poor left ventricular function as the cause for pulmonary edema causing refractory hypoxemia
Reconnect to a ventilator after ensuring there is no ventilator malfunction	Gives additional information on the patient's respiratory mechanics such as compliance and resistance
Connect to an $EtCO_2$ monitor, whenever available	Helps in confirming the tracheal placement of endotracheal tube and diagnosing additional pathologies such as bronchospasm, endobronchial intubation, circuit leaks, and large pneumothorax
Perform proning and recruitment maneuvers and PEEP adjustment if ARDS is suspected	ARDS is the most common cause of refractory hypoxemia in ICU
Use of EIT	Whenever available use of EIT will help diagnosis whether the patient has ventilation defect or a perfusion defect or ventilation-perfusion (V/Q) mismatch
Assessment of ventilator scalars and loops	Helps identify whether there is compliance or resistance issues, presence of leaks, asynchronies, auto-PEEP, etc.
Use of esophageal pressure monitoring	Helps to differentiate chest wall or abdominal compliance issues contributing to hypoxemia, adjustment of driving pressure, and detection of asynchronies
Perform bubble ECHO	Detects intracardiac shunt as a cause for refractory hypoxemia
Use of co-oximeter	Helps detect carboxy hemoglobin or methemoglobin as causes of refractory hypoxemia in suspected poisoning cases

(A-a: alveolar-arterial; ARDS: acute respiratory distress syndrome; BLUE: bedside lung ultrasonography in emergency; COPD: chronic obstructive pulmonary disease; $EtCO_2$: end-tidal carbon dioxide; EIT: electrical impedance tomography; ICU: intensive care unit; $PaCO_2$: partial pressure of carbon dioxide; P/F: partial pressure of oxygen (PaO_2)/fraction of inspired oxygen (FiO_2); PEEP: positive end expiratory pressure)

Initial Approach to the Patient with Refractory Hypoxia

Despite receiving treatment, the patient still has significant hypoxia.

Following are the causes of refractory hypoxemia:

- Potential misses or additional mechanisms for hypoxia.
  - It may be too early for improvement.
  - Rule out common causes, e.g., pneumothorax, collapse of lung segments.
  - Evaluate for additional mechanisms (e.g., severe ARDS with reversal of cardiac shunt; pneumonia with pulmonary embolism).
- Causes with less optimized therapies, e.g., ARDS is a common cause for refractory hypoxia.
- *Less common causes:* Ventilation/perfusion (V/Q) mismatch, intracardiac shunts, venous malformations, etc. **(Table 2)**.

Most important causes for refractory hypoxemia are due to one of or combination of the following pathophysiological mechanisms at the bedside irrespective of etiology such as (1) Intrapulmonary right-to-left shunting brought on by acute lung injury, ARDS, and pulmonary edema; (2) V/Q mismatch brought on by pulmonary embolism, atelectasis, pulmonary edema, and lung infiltrates like pneumonia; (3) Hypoventilation resulting from malfunctions, leaks, or errors in calibration; (4) Intracardiac shunts, or opening of patent ovale; and (5) Other reasons include anemia, reduced cardiac output, and situations of elevated demand **(Flowchart 1)**. It is imperative to evaluate for such causes and look for clinical signs of diseases that may lead to such conditions.

How to Use Adjuncts such as EIT, ABG, and Ultrasound at the Bedside to Determine the Pathophysiological Mechanism Causing Hypoxemia in Mrs N?

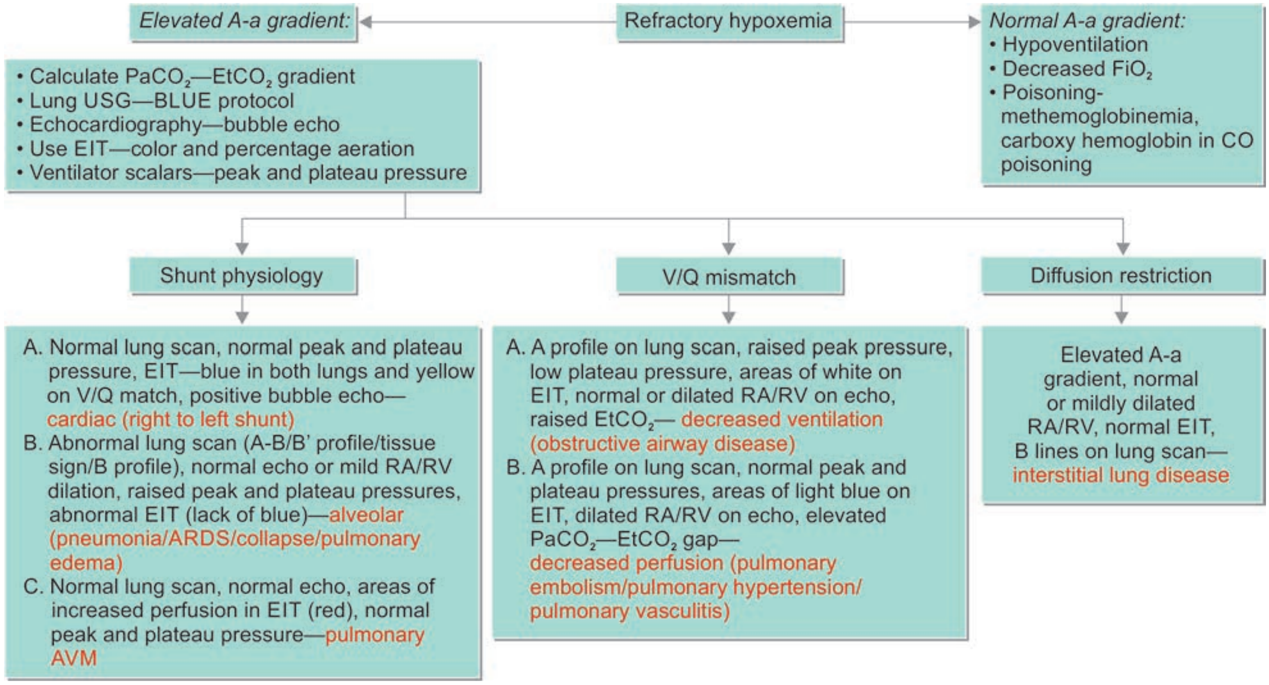
The arterial blood gas (ABG) helps in calculating the severity of hypoxemia using the oxygenation index (OI), partial pressure of carbon dioxide ($PaCO_2$)-end-tidal carbon dioxide ($EtCO_2$) gap and P/F ratio. OI is calculated using the formula OI = MAP × FiO_2 × $100/PaO_2$, where

TABLE 2: Causes of acute refractory hypoxemia in intensive care unit.

Pulmonary	***ACHD***	***Other rare causes***
ARDS	Patent foramen ovale	Acute eosinophilic pneumonia
Acute pulmonary embolism	Atrial or ventricular septal defect	Acute hypersensitivity pneumonitis
Acute pulmonary edema	Ebstein's anomaly	Drugs toxicity such as amiodarone, methotrexate, bleomycin, targeted therapy, and immune-checkpoint inhibitors
Acute exacerbation of bronchial asthma/COPD	Tetralogy of Fallot	Acute interstitial pneumonia (Hamman–Rich syndrome)
Tension pneumothorax	Single ventricle	Malignancy such as lymphangitic carcinomatosis, lung adenocarcinomas, and acute myeloid leukemia
Foreign body aspiration	Pulmonary or tricuspid atresia	Poisoning due to carbon monoxide or methemoglobinemia
Diffuse alveolar hemorrhage	Transposition of the great arteries	Air or gas embolism
Acute exacerbation of interstitial lung disease	Common arterial trunk	Arteriovenous malformations due to hereditary hemorrhagic telangiectasia, trauma, tumors, and amyloidosis
	Eisenmenger syndrome	Hepatopulmonary syndrome

(ACHD: adult congenital heart disease; ARDS: acute respiratory distress syndrome; COPD: chronic obstructive pulmonary disease)

Flowchart 1: Explaining arrival of pathophysiological diagnosis using adjuncts such as USG, ECHO, EIT, and ABG.


Refractory hypoxemia
Elevated A-a gradient:
• Calculate $PaCO_2$—$EtCO_2$ gradient
• Lung USG—BLUE protocol
• Echocardiography—bubble echo
• Use EIT—color and percentage aeration
• Ventilator scalars—peak and plateau pressure
Normal A-a gradient:
• Hypoventilation
• Decreased FiO_2
• Poisoning-methemoglobinemia, carboxy hemoglobin in CO poisoning
Shunt physiology
V/Q mismatch
Diffusion restriction
A. Normal lung scan, normal peak and plateau pressure, EIT—blue in both lungs and yellow on V/Q match, positive bubble echo—cardiac (right to left shunt)
B. Abnormal lung scan (A-B/B' profile/tissue sign/B profile), normal echo or mild RA/RV dilation, raised peak and plateau pressures, abnormal EIT (lack of blue)—alveolar (pneumonia/ARDS/collapse/pulmonary edema)
C. Normal lung scan, normal echo, areas of increased perfusion in EIT (red), normal peak and plateau pressure—pulmonary AVM
A. A profile on lung scan, raised peak pressure, low plateau pressure, areas of white on EIT, normal or dilated RA/RV on echo, raised $EtCO_2$— decreased ventilation (obstructive airway disease)
B. A profile on lung scan, normal peak and plateau pressures, areas of light blue on EIT, dilated RA/RV on echo, elevated $PaCO_2$—$EtCO_2$ gap— decreased perfusion (pulmonary embolism/pulmonary hypertension/ pulmonary vasculitis)
Elevated A-a gradient, normal or mildly dilated RA/RV, normal EIT, B lines on lung scan— interstitial lung disease

(A-a: alveolar-arterial; ABG; arterial blood gas; ARDS: acute respiratory distress syndrome; AVM: arteriovenous malformation; BLUE: bedside lung ultrasonography in emergency; CO: carbon monoxide; echo: echocardiogram; EIT: electrical impedance tomography; $EtCO_2$: end-tidal carbon dioxide; FiO_2: fraction of inspired oxygen; $PaCO_2$: partial pressure of carbon dioxide; RA: right atrium; RV: right ventricle; USG: ultrasonography; V/Q: ventilation-perfusion)

MAP indicates mean airway pressure and FiO_2 indicates the fraction of inspired oxygen. Hypoxemia is classified based on OI as shown in **Table 3**.

The P/F ratio is more commonly accepted for severity classification in ARDS.

An elevated $PaCO_2$-$EtCO_2$ gap is suggestive of V/Q mismatch or dead space issues. It can be seen in pulmonary embolism or severe ARDS (due to microthrombi in pulmonary circulation). Alveolar-arterial gradient is normal in cases of hypoventilation/decreased FiO_2 as

TABLE 3: Classification of hypoxemia based on oxygenation index.

<8	Mild
8–16	Moderate
16–40	Severe
≥40	Very severe

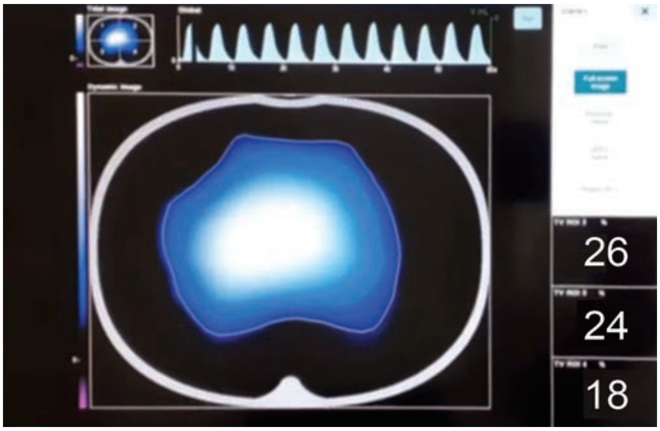


Fig. 1: Well-ventilated areas in both lungs in electrical impedance tomography (EIT).

a cause for hypoxemia whereas it is elevated in all other causes of hypoxemia.

Following bedside lung ultrasonography in emergency (BLUE) protocol, one can arrive at diagnosis which explains the cause for hypoxemia (e.g., shunt physiology in pneumonia, lung collapse, or ARDS). Normal lung scan should immediately prompt an echocardiographic evaluation. For further details refer to Chapters 15 and 26.

The EIT is a new noninvasive lung imaging technology that also helps detect abnormality at the patient's bedside. After being positioned around the patient's thorax in one transverse plane (usually fourth intercostal parasternal space), a silicone EIT belt with surface electrodes should be attached to the EIT monitor. Gray areas indicate matched units that are both well-ventilated and perfused. Areas with low ventilation are shown in dark blue, with lack of ventilation as the absence of color whereas areas with high ventilation are shown in white **(Figs. 1 and 2)**. Red and light blue markers indicate high and low perfusion, respectively not shown in the image.

Thus, EIT helps to detect most of lung pathologies such as collapse or overdistension and helps in PEEP adjustment too.

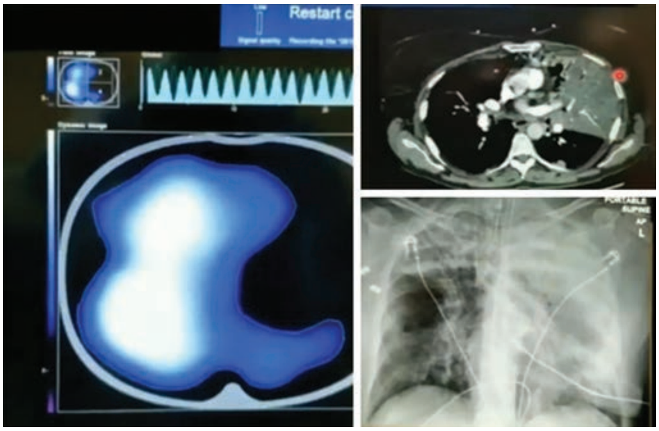


Fig. 2: A ventilation defect in left anterior region. It was corroborated on chest X-ray (CXR) and computed tomography (CT) scan which showed a left upper lobe collapse.

At the end of the above steps, clinician should come to a conclusion with regard to the pathophysiological mechanism causing refractory hypoxemia.

HISTORY AND SYMPTOMATOLOGY

History taking should focus on narrowing down the differential diagnosis to 2–4 causes of refractory hypoxemia in Mrs N. At the end of history-taking, the clinician should be able to diagnose whether the hypoxemia is cardiac or pulmonary or combined etiology. A comprehensive history can be retaken at a later time. *See* Chapters 10 and 15 for details.

Keeping the above causes in mind, history-taking should be focused on fever, cough, sputum production if any, hemoptysis, breathlessness, chest pain, and wheezing. Systemic symptoms such as weight loss, malaise, and night sweats are suggestive of tuberculosis, connective tissue disorders, or malignancy. Symptoms such as easy fatigability, palpitations, exertional dyspnea, squatting episodes, and cyanosis since childhood are more suggestive of congenital heart diseases. History of cough, chest pain, fever, and dyspnea for short duration suggest pulmonary embolism [especially in the background history of deep vein thrombosis (DVT)], whereas similar complaints since childhood along with exercise intolerance and dyspnea on upright position (platypnea) point toward pulmonary arteriovenous malformations.

GENERAL PHYSICAL EXAMINATION

General physical examination is important in patients with refractory hypoxia since it gives crucial information to identify potential causes or complications. This provides clues to identify signs of underlying systemic diseases, severity of disease, and signs of complication that add to the existing hypoxia and may guide further diagnostic tests and interventions. It is good to be systematic and elicit all signs while performing examination, refer to Chapters 10 and 15 for details. *See* **Box 1** for a summary of the general examination, only findings that are very specific to refractory hypoxia are given. Similarly, the systemic examination will be specific to the examination of patients with refractory hypoxia granted that the basic steps of examination are performed.

HOW TO PROCEED WITH THE SYSTEMIC EXAMINATION IN PATIENTS WITH REFRACTORY HYPOXEMIA?

Respiratory System

Respiratory system examination in ICU has the following components—clinical examination, lung ultrasound, assessment of ventilator parameters, interpretation of ABGs, assessment using EIT, and esophageal pressure monitoring. In refractory hypoxemia, examination should be directed to confirm the differential diagnosis arrived so far from history and troubleshooting of hypoxemia.

BOX 1: Focused general physical examination in a patient with refractory hypoxemia.

- *Vitals: Have a quick look at the vital signs and act appropriately*
- *Saturation:* Make a mental note of FiO_2 and PEEP requirements to access the degree of hypoxemia. Generally, the rule of thumb for alveolar PaO_2 is 5 times the set FiO_2. Using this, one can calculate the alveolar-arterial (A-a) gradient and PaO_2/FiO_2 (P/F) ratio at the bedside without an ABG

  For example, if the saturation is 90% for a FiO_2 of 100 and PEEP of 15, then PaO_2 is 60 (based on the oxygen dissociation curve). Then A-a gradient is 500 – 60 = 540 and the P/F ratio is 60
- P—Seen in any chronic disease
- I—Icterus can be seen in pneumonia with sepsis secondary to cholestasis/hemolysis as in cases of SLE/ANCA vasculitis/APLA syndrome
- C—Central cyanosis is commonly seen with cardiac causes of refractory hypoxemia as in **Table 2** whereas peripheral cyanosis suggests low cardiac output states or Raynaud's phenomenon. The presence of Raynaud's phenomenon should arouse a suspicion of SLE/mixed connective tissue disorder or paraneoplastic syndrome in Mrs N
- C—Clubbing can be seen in chronic parenchymal lung diseases and cyanotic heart diseases
- L—Generalized lymphadenopathy is seen in autoimmune diseases, scrub typhus, leptospirosis, or viral etiology
- E—Edema (bilateral) can be due to Cor pulmonale (acute—secondary to ARDS or pulmonary embolism; chronic—COPD/asthma or congenital heart disease). Unilateral edema should arouse a suspicion of deep vein thrombosis causing pulmonary embolism
- *Jugular venous pressure* can be elevated in cases of acute Cor pulmonale or cardiac illness
- *Skin and mucosa examination:* Check for cutaneous manifestations of autoimmune diseases such as palpable purpura, pyoderma gangrenosum, urticaria-like rashes, and necrotic ulcers
- Mucosal telangiectasias **(Fig. 3)** can be seen in hereditary hemorrhagic telangiectasia (hypoxemia could be due to pulmonary AVMs)
- Is the face flushed (seen in carbon dioxide narcosis)?
- Tropical fevers can present with the following types of rash:
  - Maculopapular—scrub typhus/typhoid
  - Petechiae/ecchymoses—rickettsioses, viral hemorrhagic fevers, yellow fever, dengue, and leptospirosis
  - Eschar—tick bite fever, scrub typhus
- The oral examination is important to check for oral ulcers (connective tissue disorders), central cyanosis (congenital heart disease), and missing tooth or dentures (foreign body aspiration)

  Beddogram
- *Devices*—reassess tube position and ventilator scalars and loops. Check for the presence of additional devices like intercostal drainage (ICD) or pigtail catheter. If there is an ICD, check for column movement or bubbling in the underwater seal (indicates bronchopleural fistula). If there is no column movement ensure that ICD is not migrated outside of the intrathoracic location. Check ventilator circuit is not kinked or clamped. Is there any low tidal volume or low Ppeak alarm? Could there be any disconnection or ventilator malfunction? If EIT is attached, is there any ventilation or perfusion defect? Is there any V/Q mismatch?

(ABG: arterial blood gas; ANCA: antineutrophil cytoplasmic antibody; APLA: antiphospholipid antibody; ARDS: acute respiratory distress syndrome; AVM: arteriovenous malformations; COPD: chronic obstructive pulmonary disease; EIT: electrical impedance tomography; FiO_2: fractional inspired oxygen; ICD: intercostal drainage; PaO_2: partial pressure of oxygen; PEEP: positive end-expiratory pressure; SLE: systemic lupus erythematosus; V/Q: ventilation-perfusion)

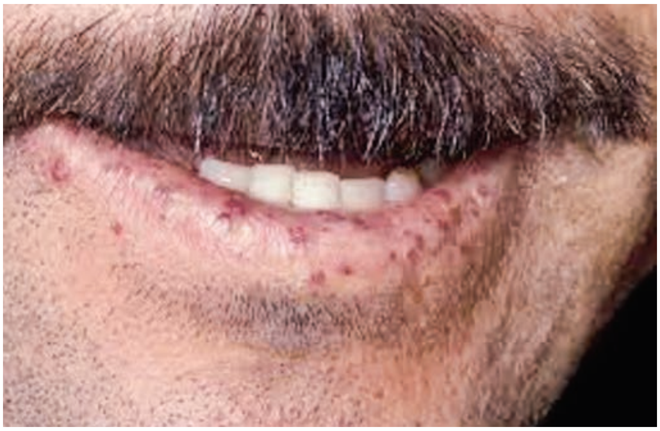


Fig. 3: Mucosal telangiectasias.
Source: Wikipedia. Hereditary hemorrhagic telangiectasia. [online] Available from https://en.wikipedia.org/wiki/Hereditary_hemorrhagic_telangiectasia. [Last accessed August, 2024].

Note: At each step rule out the common causes that are missed and technical errors. Then examine for specific causes of refractory hypoxia.

Inspection

- On inspection, note the appearance of the chest wall. Check for asymmetry of the chest.
- *Endotracheal tube:* Check the tube size and fixation length. Is it appropriate? Or has the tube migrated proximally or distally? Are there any secretions blocking the tube?
- *Heat moisture and exchange (HME) filter:* Is it blocked?
- *$EtCO_2$:* Analysis of $EtCO_2$ waveform can detect bronchospasm, leaks, and circuit disconnection.
- EIT images to look for defects in ventilation, perfusion, or V/Q mismatch.
- *Esophageal pressure monitoring:* To calculate transpulmonary pressures and optimize PEEP. It can also be used to identify ineffective efforts due to dynamic hyperinflation and auto-PEEP in chronic obstructive pulmonary disease (COPD)/asthma.

Palpation

- Palpate for crepitus (indicates subcutaneous emphysema which may be due to pneumothorax) and enlarged lymph nodes.
- Check for the intactness of the cuff and measure cuff pressure using a manometer.
- Check for any small circuit leaks, especially if the volume time scalar is not reaching baseline.

Auscultation

A clear chest should arouse the suspicion of pulmonary embolism and lack of breath sounds unilaterally indicates a tension pneumothorax or lung collapse.

Adjuvants to Examination

Lung Ultrasound

The BLUE protocol must be followed in all hypoxemic patients. It helps diagnose the cause of refractory hypoxemia with sensitivity and specificity comparable to that of computed tomography. It is quick and easy to perform. The learning curve is not steep. Remedial life saving measures can be instituted early to treat the life-threatening hypoxemia based on these findings. All these make lung ultrasound an indispensable tool for management of patients with refractory hypoxemia.

A detailed description of BLUE protocol and lung ultrasonography is given elsewhere. *See* Chapter 15.

Assessment of Ventilator Scalars and Outputs

- *Step 1:* Make FiO_2—100%
- *Step 2:* Follow scalar assessment of ventilator during emergencies (SAVE) protocol **(Flowchart 2)**

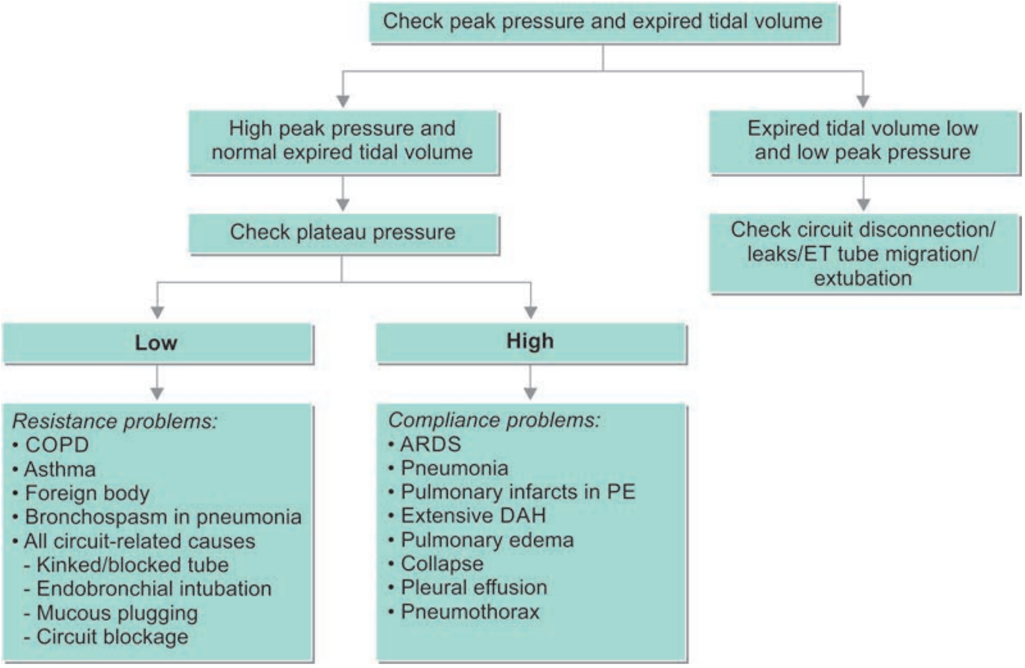
Interpretation of Arterial Blood Gases

It is important to get carboxy hemoglobin or methemoglobin in suspected poisoning cases. Comprehensive assessment of ABG is needed in refractory hypoxemia which includes calculating P/F ratio, OI, alveolar-arterial (A-a) gradient, and $PaCO_2$-$EtCO_2$ gap. The use of ABG to arrive at a diagnosis is already explained in **Flowchart 1**.

Assessment Using Adjuncts such as Electrical Impedance Tomography and Esophageal Pressure Monitoring

The next step in the approach to hypoxemia includes the use of available adjuncts such as EIT and esophageal pressure monitoring. The use of EIT has already been described. Esophageal pressure (Pes) monitoring is valuable in patients with COPD/asthma to detect asynchronies such as in effective efforts **(Fig. 4)** and delayed cycling when patients are on pressure support.

Flowchart 2: SAVE protocol—scalar assessment of ventilator during emergencies.


Check peak pressure and expired tidal volume
High peak pressure and normal expired tidal volume
Expired tidal volume low and low peak pressure
Check plateau pressure
Check circuit disconnection/ leaks/ET tube migration/ extubation
Low
High
Resistance problems:
• COPD
• Asthma
• Foreign body
• Bronchospasm in pneumonia
• All circuit-related causes
- Kinked/blocked tube
- Endobronchial intubation
- Mucous plugging
- Circuit blockage
Compliance problems:
• ARDS
• Pneumonia
• Pulmonary infarcts in PE
• Extensive DAH
• Pulmonary edema
• Collapse
• Pleural effusion
• Pneumothorax

(ARDS: acute respiratory distress syndrome; COPD: chronic obstructive pulmonary disease; DAH: diffuse alveolar hemorrhage; ET: endotracheal; PE: pulmonary embolism)

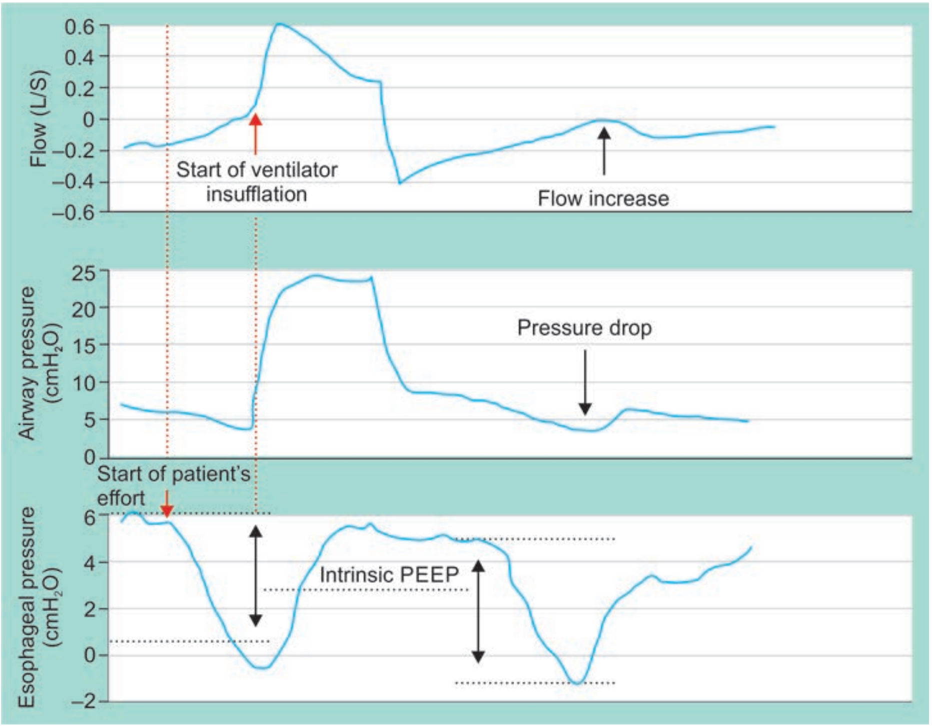


Fig. 4: Ineffective triggers due to auto-PEEP. (PEEP: positive end-expiratory pressure)

WHAT ARE THE BENEFITS OF ESOPHAGEAL PRESSURE MONITORING IN PATIENTS WITH REFRACTORY HYPOXIA (E.G., ARDS)?

Calculating inspiratory and expiratory transpulmonary pressures is helpful, especially in ARDS patients who are obese (chest wall compliance can be calculated) and pancreatitis (intra-abdominal hypertension or pleural effusion affecting chest wall compliance). As chest wall compliance decreases, pleural pressure rises. In these patients, there may be areas of derecruitment due to elevated pleural pressures which may be adding to the existing hypoxemia. Maintaining an end-expiratory transpulmonary pressure of zero in these patients and adjusting PEEP accordingly may help alleviate derecruitment and improve oxygenation. Clinicians can also use a transpulmonary pressure target below 20 cm of water to optimize PEEP at the bedside.

In ARDS, limiting transpulmonary pressures below 20 cm of water prevents ventilator-induced lung injury (VILI). Also, limiting tidal changes in transpulmonary pressures to <10–12 cm of water is advised to prevent VILI in ventilated patients and patient self-inflicted lung injury (P-SILI) in patients with spontaneous efforts. And it should be below 25 cm of water during recruitment maneuvers. All these are possible only with monitoring of esophageal pressures.

With Pes, one can also calculate chest wall, lung compliance, and respiratory system compliance **(Fig. 5)**.

Esophageal pressure measured at lower one-third is a surrogate of pleural pressure. Thus, esophageal pressure monitoring helps in PEEP adjustment, calculation of transpulmonary pressure, and hence monitoring of lung stress and VILI, and also in monitoring ventilator asynchronies.

Inspiratory and expiratory transpulmonary pressures can be calculated as follows:

$$P_{\text{transpulmonary}} = P_{\text{airway pressure}} - P_{\text{pleural pressure}}$$

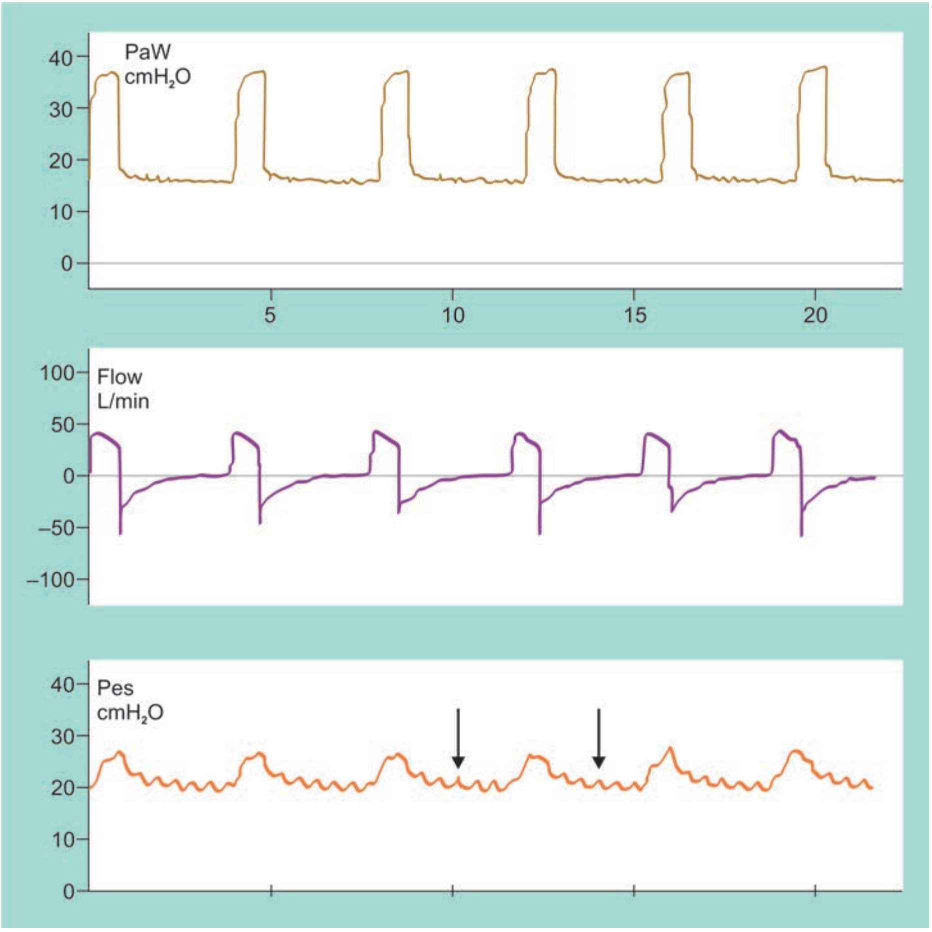


Fig. 5: Esophageal pressure monitoring. The arrows suggest cardiac oscillations confirming the position of the catheter.

REFRACTORY HYPOXEMIA: CASE SCENARIO (TABLE 4)

TABLE 4: Focused history-taking and examination in patients with refractory hypoxia.

Case scenarios: There are four hypothetical scenarios as examples. The main context of the case is kept the same in all four scenarios, and few modifications were made to the scenario to discuss the case-specific details while examining patients with refractory hypoxia.

Hypothetical scenario What to ask and look for in patients with (refractory) hypoxia a short-focused guide	Hypothetical scenario-A	Hypothetical scenario-B	Hypothetical scenario-C	Hypothetical scenario-D
First information	Mrs M, a 28-year-old female farmer, was admitted to the ICU with 5 days fever, cough, and 2 days breathlessness. She is a farmer growing paddy with no prior significant medical history. She received treatment from primary health center treated with third generation cephalosporin, doxycycline, paracetamol, oxygen, and IV fluids. Given worsening breathlessness, she was referred to your hospital. In the emergency department, she is intubated given impending respiratory failure and shifted to ICU. Currently, she is intubated, sedated, and paralyzed. Her oxygen support includes FiO_2 at 100% and PEEP at 12 cmH_2O. After 8 hours of ICU treatment, her SpO_2 is 93%, and the P/F ratio is 90	Mrs N, a 38-year-old female, who is obese, a known case of bronchial asthma (BA), rheumatoid arthritis on oral steroids was admitted to the ICU with 5 days fever, cough, and 2 days breathlessness. She received treatment from primary health center treated with third generation cephalosporin, doxycycline, paracetamol, oxygen, and IV fluids. She has frequent hospital admissions for BA. Given worsening breathlessness, she was referred to your hospital. In the emergency department, she was intubated given impending respiratory failure and shifted to ICU. Currently, she is intubated, sedated, and paralyzed. Her oxygen support includes FiO_2 at 100% and PEEP at 12 cmH_2O. After 8 hours of ICU treatment, her SpO_2 is 93%, and the P/F ratio is 90	Mrs Y, a 31-year-old female, with no known comorbidities, was admitted to the ICU with 5 days fever, cough, and 2 days breathlessness. She received treatment from primary health center treated with third generation cephalosporin, doxycycline, paracetamol, oxygen, and IV fluids. The father informed that she visited nearby clinic for sore throat twice this month. Also, she feels more tired and is not eating well. Given worsening breathlessness, she was referred to your hospital. In the emergency department, she was intubated given impending respiratory failure and shifted to ICU. Currently, she is intubated, sedated, and paralyzed. Her oxygen support includes FiO_2 at 100% and PEEP at 12 cmH_2O. After 8 hours of ICU treatment, her SpO_2 is 93%, and the P/F ratio is 90	Mrs Z, a 24-year-old female, has history of wheezing when exposed to dust and smoke, was admitted to the ICU with 5 days fever, cough, and 2 days breathlessness. She received treatment from primary health center treated with third generation cephalosporin, doxycycline, paracetamol, oxygen, and IV fluids. Given worsening breathlessness, she was referred to your hospital. In the emergency department, she is intubated given impending respiratory failure and shifted to ICU. Currently, she is intubated, sedated, and paralyzed. Her oxygen support includes FiO_2 at 100% and PEEP at 12 cmH_2O. After 8 hours of ICU treatment, her SpO_2 is 93%, and the P/F ratio is 90. The mother gave history of sudden blackouts and her lips and fingers turned blue when she had severe wheezing episode 6 months back requiring short hospital admission

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History and symptomatology (what to specifically ask for and what our patients had). These are some common information the physician should enquire for, not limited to the list provided. Examples of focused history taking respect to hypoxia (refractory).				
• Work and travel history (garden, travel to area with bushes, trekking, etc.) • Activities of daily living, recent body weight changes, appetite, sleep • Recent frequent infections, admissions, medications • Cyanosis, stroke, syncope, etc. • Swellings (lymph node), rash, ulcers (Eschar) • *Progression of symptoms (any tropical fever):* Temporal relations • Any significant past history (exacerbations of preexisting diseases, response to treatment, etc.)	• Gardener, exposure to grass and bushes • Fever, followed by cough and later breathlessness • Had leg pain, sweating, headache, and palpitations	• H/O of recent changes in weight—weight increased • Altered sleep patterns and snoring • *Medication history:* On oral steroids and inhalers • *Frequency of exacerbations:* Once in 2 months • Treatment response (resolving with steroids, noncompliant to treatment) • No previous history of mechanical ventilation or tracheostomy • No H/O exacerbation of rheumatoid arthritis. No systemic involvement (Remember ILD as one of differentials in long-standing RA patients)	• Has H/O night sweats, excessive sweating, and easy fatigability • Has some lumps or swellings noticed by family near the neck • *Recent clinic visit:* The records showed H/O recurrent sore throats or infections, treated with analgesics and antibiotics • Family noticed changes in appetite or abdominal discomfort	• History of turning blue • Episodes of syncope or near-syncope, TIA, or stroke • Exercise tolerance before current illness • No history of cardiorespiratory disease in family or siblings
General physical examination (what to specifically look for and what our patients had). These are some common information/general examination findings the physician should enquire for, not limited to the list provided. Examples of focused examination.				
• Facies, facial congestion, eye signs • *Oral examination:* Candidiasis, tonsilitis, ulcers • Rashes, Eschar conjunctivitis/conjunctival hemorrhage (leptospirosis) • Body habitus (obesity), abdominal distension, COPD • Lymphadenopathy • Differential limb swelling/edema	• Flushed trunk and face—sepsis • *Mild cervical lymphadenopathy:* Infections • *Eschar in inguinal region:* Scrub typhus • *Petechiae, ecchymosis:* Dengue • *Icterus:* Sepsis cholestasis, icteric leptospirosis	• *BMI:* 42 kg/m^2 (morbidly obese) • *Skin:* Striae, moon facies, buffalo hump suggestive of Cushing's syndrome • Noticed central obesity • *Peripheral edema:* Pitting (fluid) versus not-pitting (thyroid, Cushing)	• Flushed neck and face, prominent neck veins, swelling of face noticed compared to lean body • SVC compression/high intrathoracic pressure • Right upper limb edema • *Generalized lymphadenopathy*: Cervical, axillary, and inguinal (soft to firm and nontender) enlarged tonsils • *Signs of recent weight loss:* Striae in abdomen	Central cyanosis, (mixing of venous blood)—cyanosis should trigger evaluation for cardiac etiology

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Systemic examination (Cardiorespiratory): Focused and specific examination findings to look for in patients with refractory hypoxia, not limited to the list provided.				
Inspection to auscultation Refer Chapters 10 and 15 • $EtCO_2$ • $PaCO_2$-$EtCO_2$ gap (dead space, pulmonary blood flow) • Ventilator graphics and screen • *POCUS:* Lung and heart, bubble Echo in selective cases • EIT—if present • *Esophageal pressure:* Selected cases (ARDS) if available	*Clinical examination:* Chest movements appear symmetrical, reduced air entry left intra-axillary, crepitation both sides *Ventilator screen:* • High peak airway pressures (34 cmH_2O) noted on ventilator • Pressure time scalar—longer initial part of scalar (affected by resistance) and minimally altered slope (affected by compliance), reduced peak expiratory flow in flow time scalar (reflect high resistance) • If plateau pressure is rising, suspect dynamic hyperinflation or pneumothorax • End-tidal CO_2 waveform shows prolonged expiratory phase	*Clinical examination:* Chest movements appear symmetrical, reduced air entry left intra-axillary, and crepitation both sides *Ventilator measurements:* • High plateau pressures (35 cmH_2O) • Low compliance (25 mL/cmH_2O) • PEEP set at 12 cmH_2O • Driving pressure (13 cmH_2O) Esophageal pressure *End-inspiration:* • Paw (peak airway pressure) = 35 cmH_2O • Pes (esophageal pressure) = 15 cmH_2O *End-expiration:* • Paw (PEEP) = 12 cmH_2O • Pes (esophageal pressure) = 15 cmH_2O *Transpulmonary pressure:* *Inspiration:* +20 (risk of overdistension), *Expiration:* –3 (risk of atelectasis) • EIT shows dorsal lung collapse and heterogeneous ventilation	*Clinical examination:* Reduced chest expansion on the right side, fullness of right side present, and dull on percussion. Reduced breath sounds of both sides, right (R) > left (L) • High airway pressures (peak 42 cmH_2O, plateau 31 cmH_2O) • Flow-volume loop showing increased inspiratory resistance • Prolonged expiratory phase on capnography	*Clinical examination:* Chest symmetrical. No significant abnormalities • Systolic murmur, parasternal heave • *Echocardiography:* Right ventricular dilation, elevated pulmonary artery pressures • *Bubble study:* Positive, confirming right-to-left shunt
Comments	*Potential diagnosis:* • Tropical fever with ARDS, endobronchial intubation aggravating the hypoxia • The hypoxia may not be so refractory, the endobronchial intubation was additive to worsen the hypoxia • Prone ventilation may help to improve oxygenation	*Potential diagnosis:* Acute exacerbation of bronchial asthma *Possible Cushing's syndrome:* Obesity (increased chest wall elastance) *Causes for persistent hypoxia in this case* • Suboptimal PEEP • ARDS	*Potential diagnosis:* (Hodgkin's) Lymphoma with community-acquired pneumonia, SVC compression Mediastinal mass compressing on right bronchus (evident due to paralytic agents since the patient lost muscle tone after intubation) Obstruction and parenchymal problem	*Potential diagnosis:* Patent foramen ovale. Worsening hypoxia when there is high right atrial pressure (Acute cor pulmonale or PHTN with ARDS). Can cause cyanosis when severe attack and lead to paradoxical embolism in special situations

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			Prone positioning can help in case of anterior mediastinal mass which is not adherent and only compressing on bronchus. The mass will fall forward relieving some airway obstruction and in turn will improve hypoxia	
Potential causes of refractory hypoxia in each case.	*Potential misses for hypoxia:* Endobronchial intubation.	*Causes with less optimized therapies:* The alveoli collapsed due to suboptimal PEEP	*Additional mechanisms for hypoxia:* ARDS + airway compression	*Additional mechanisms for hypoxia:* ARDS + PFO

(ARDS: acute respiratory distress syndrome; BMI: body mass index; cmH_2O: centimeters of water; COPD: chronic obstructive pulmonary disease; Echo: echocardiography; EIT: electrical impedance tomography; EtCO_2: end-tidal carbon dioxide; FiO_2: fractional inspired oxygen; H/O: history of; ICU: intensive care unit; ILD: interstitial lung disease; IV: intravenous; P/F: PaO_2/FiO_2; PaCO_2: partial pressure of carbon dioxide; PaO_2: partial pressure of oxygen; PEEP: positive end-expiratory pressure; PFO: patent foramen ovale; PHTN: pulmonary hypertension; POCUS; point-of-care ultrasonography; RA; rheumatoid arthritis; SpO_2: oxygen saturation; SVC: superior vena cava; TIA: transient ischemic attack)

Replacing pleural pressure with esophageal pressure in the equation,

$P_{\text{transpulmonary}}\,(P_{\text{tm}}) = P_{\text{airway pressure}} - P_{\text{esophageal pressure.}}$

When all measured at the end-inspiration,

Inspiratory $P_{\text{tm}} = P_{\text{plat}} - \text{Pes}$. ($P_{\text{plat}}$ – Plateau pressure)

When measured at end-expiration,

Expiratory $P_{\text{tm}} = \text{PEEP} - \text{Pes}$

The PEEP should be assessed by performing an expiratory hold maneuver to avoid errors in cases of coexisting auto-PEEP due to high rates of ventilation.

CARDIAC EXAMINATION

A focused examination of cardiovascular system should include inspecting for any surgical scars (previous cardiac surgeries) and precordial pulsations. Screening echocardiography identifies all valvular lesions and right to left shunt especially Eisenmenger syndrome should be identified. A bubble echo helps in identifying intracardiac shunts.

CASE SCENARIO PROGRESS

On further review of history, Mrs N had history of H/O of the recent increase in body weight in the past 6 months, gained 8 kg, and altered sleep and snoring. She is on oral steroids and inhalers for her asthma and rheumatoid arthritis. On examination, she is morbidly obese, has moon facies, and edematous legs. She is ventilated on assist control mode with fentanyl and propofol infusion **(Table 4)**. There are four hypothetical scenarios as examples. The main context of the case is kept the same in all four scenarios, and few modifications were made to the scenario to discuss the case-specific details while examining patients with refractory hypoxia **(Table 4)**. It provides a summary of eliciting focused history and examination of patients with refractory hypoxia.

SUMMARY

It is an art to elicit a focused history and examination. This approach is very helpful when rapidly examining patients with refractory hypoxia. A systematic approach involving eliciting history, clinical examination, assessment of ventilator scalars, use of the SAVE protocol, lung ultrasound, bubble echocardiography, and interpretation of ABGs are essential steps to arrive at a bedside diagnosis in a patient with refractory hypoxemia. EIT and esophageal pressure monitoring provide valuable insights and help troubleshoot refractory hypoxemia, especially in ARDS patients.

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Chapter

35

How will I Approach/Examine a Patient with Oliguria?

Abhishek Samprathi, Manu Varma MK, Natesh Prabu R

ABSTRACT

Oliguria, a state of low urine output, is often a presenting symptom/sign or associated with acute kidney injury. It is crucial to aggressively evaluate and treat it to prevent its progression to acute kidney injury, which is associated with high morbidity and mortality. Careful stepwise clinical examination combined with a bedside point-of-care ultrasound (POCUS) is beneficial in the assessment of oliguria and can also be used to monitor treatment response. This chapter gives an overview of the examination of a patient who presents with oliguria.

INTRODUCTION

Oliguria essentially is low urine output due to any reason. It is important to understand that oliguria can be physiological or pathological. It is a reflector of kidney injury, most often acute renal injury. The intensivists often come across patients with renal injury in patients admitted to the intensive care unit (ICU) and many a time the cause is multifactorial. Commonly, oliguria is the trigger for further evaluation of the cause and serves as a monitoring tool in patients with circulatory failure and renal injury to know the recovery. A careful history-taking and clinical examination of patients with oliguria/kidney injury gives a clue to the diagnosis. The examination of the renal system can be preplanned as a part of daily ICU rounds or on demand. The examination should be focused, rapid, and systematic. In this chapter, we will be discussing different methods to approach a patient with oliguria.

CASE SCENARIO

A 46-year-old gentleman has presented with a few days history of general malaise, fever, back pain, and decreasing urine output. He had passed very little "dark yellow" colored urine yesterday. He appeared to be anxious and tachypneic. On examination, he had tenderness in the right flank. On auscultation, fine crepitations were heard over lung bases. How would you further evaluate this patient and manage it?

The above history and examination are suggestive of acute kidney injury (AKI) with oliguria, possibly secondary to urosepsis. This brief discussion will discuss a stepwise clinical approach to oliguria and its differential diagnosis.

History and Symptomatology

It is always important to review a detailed history, which could give us further clues for the diagnosis. On further questioning, he revealed passing dark urine over the past few days with gradually smaller amounts being passed in the last 2 days. He had not passed any urine in the last 24 hours. He did not have any comorbidities and was not on any medication. He had no family history of renal illness. However, he had noticed that he had leg swelling.

Dark-colored urine could be due to various causes. Decreased urine output with <400 mL or <6 mL/kg body weight over 24 hours is defined as oliguria. However, it is important to note that oliguria and AKI are not the same. Oliguria literally means "reduced" urine volume—less than that is needed to remove the end products of metabolism. Normal kidneys can concentrate urine and excrete solutes by increasing their specific gravity. In AKI, the glomerular filtration rate is abruptly reduced, causing sudden retention of metabolites such as urea, potassium, phosphate, sulfate, creatinine, and renally excreted drugs that are normally cleared by the kidneys. The urine volume is usually <400 mL/day. If renal concentrating mechanisms are impaired, the daily urine volume may be normal or even high (*high-output or nonoliguric renal failure*). In extreme cases, anuria might be observed (urine output <100 mL/day).

Other relevant symptomatology histories to be reviewed are summarized in **Table 1**.

TABLE 1: History and symptomatology in patients with oliguria or renal injury.

S. No.	*Domain*	*Relevant history to note*	*Comments*
1.	Urine characteristics	*Urine volume:* • Less quantity of concentrated volume • Gradual reduction of volume daily • Sudden stoppage *Color and content:* Any change in color—white, yellow to dark yellow or brown, red, green, etc. **(Figs. 1 and 2)** *Urine examination:* Casts/sediments—WBC/RBC/hyaline/waxy *Associated symptoms:* • Pain while passing urine—dysuria • Low urine stream • Dribbling of urine, long time to empty the bladder • Incontinence, nocturia, polyuria • Difficulty in voiding	The information on the urine output is very important as it gives clues about the diagnosis. Patients with prerenal causes for oliguria/renal injury usually will have a gradual drop in urine output and a gradual change of urine colure (darker urine over time). Patients with intrinsic renal injury may have an altered urine color (cola color—glomerulonephritis, brown—rhabdomyolysis, hemolysis). Associated symptoms should raise suspicion of urinary tract infection, renal stones, and prostate hypertrophy
2.	Temporal events	• *Current/recent illness:* – *Acute febrile illness:* Fever, myalgia, joint pain, thrombocytopenia – Loose motions – Shock/hypotension – Recent hospitalization and medications – MI, stroke, vascular, etc. – Heatstroke – Thirst • *Recent surgery or instrumentation:* – Major surgery (cardiothoracic, abdominal, renal, etc.) – Intraoperative events (hypotension, blood transfusion, etc.) • *Recent trauma:* RTA, crush injuries, renal injury, and electrical injuries • *Medications:* Anaphylaxis, contrast administrations, NMS, and MH	The temporal events are very important. Most of the times those are causes of renal injury and oliguria. It is common to have oliguria following volume loss, hypotension, and surgery/trauma. Sepsis and tropical infections can cause a combination of prerenal and intrinsic renal injuries
3.	Comorbid conditions	• DM, HTN • CKD • CAD, DCM/HF, valvular heart disease • Autoimmune, CTDs • Urological	Check whether the comorbidities are controlled. Patients may have less renal reserve and baseline function may be less
4.	Drugs and medication	• *Nephrotoxic drugs:* – Antibiotics – NSAIDs – ACE-I, ARBs • Contrast • Blood products	Nephrotoxic drugs should be immediately stopped. Contrast-induced nephropathy can be prevented by IV fluid administration before contrast administration
5.	Family history	Renal failure in family members, first-degree relatives, and consanguineous marriage	There are certain renal diseases/ syndromes that run-in families, e.g., ADPKD, Alport syndrome, Gitelman syndrome, etc.

(ACE-I: angiotensin converting enzyme inhibitor; ADPKD: autosomal dominant polycystic kidney disease; ARB: angiotensin receptor blocker; CAD: coronary artery disease; CKD: chronic kidney disease; CTD: connective tissue disease; DCM: dilated cardiomyopathy; DM: diabetes mellitus; HF: heart failure; HTN: hypertension; MI: myocardial ischemia; MH: malignant hyperthermia; NMS: neuroleptic malignant syndrome; NSAIDS: nonsteroidal anti-inflammatory drugs; RBC: red blood cell; RTA: road traffic accident; WBC: white blood cell)

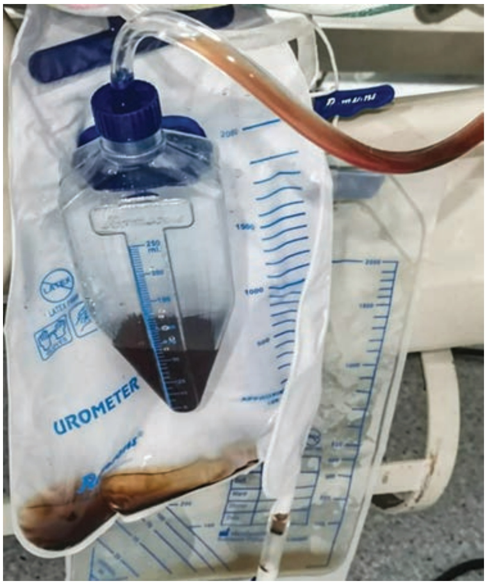


Fig. 1: Dark brown colored urine suggestive of acute tubular necrosis or rhabdomyolysis.

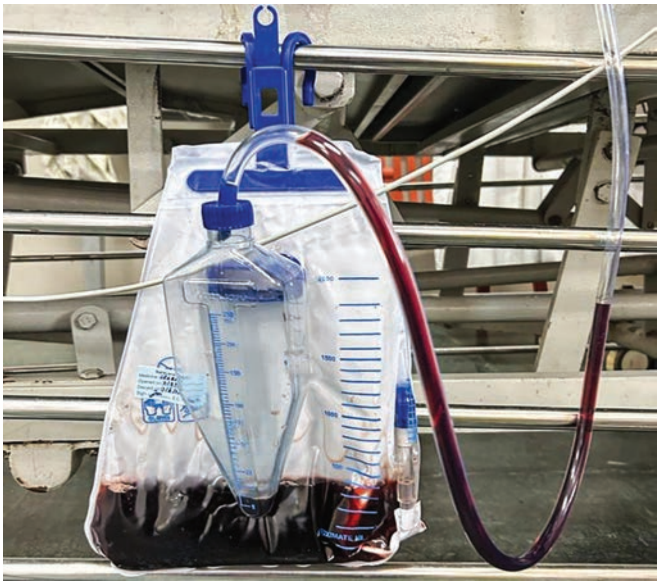


Fig. 2: Gross hemoglobinuria may appear as reddish urine when there is marked intravascular hemolysis and exceeding the renal threshold for hemoglobin.

Clinical Examination of the Patient

General Physical Examination

General examination should be quick, focused, and renal specific. To begin with, look at the vitals from the multipara monitor and intervene if there are any cardiorespiratory compromises. Rapid head-to-toe examination including bed space eliciting specific signs will give important clues to the causes of oliguria and renal injury. The examination should focus on finding the probable cause of oliguria/renal injury (prerenal, renal, or postrenal cause), and differentiate between acute and chronic kidney injury **(Table 2)**.

Positive historical findings should be followed up with a focused physical examination. Inadequate perfusion is one of the common, important, and potentially reversible causes of oliguria in ICU patients. Hence, they should be examined for other signs of inadequate perfusion such as altered mentation and capillary refilling time. It has been shown that normalization of capillary refilling time correlates with lactate clearance (Surviving Sepsis Guidelines 3). Other signs of sepsis such as fever, tachycardia, and localizing symptoms should also be evaluated.

Prerenal Kidney Injury

"Prerenal" kidney injury happens commonly due to inadequate renal perfusion due to any cause. This can be inadequate intravascular volume or a "pump failure". The renal perfusion is compromised in either of the case. Systemic congestion also leads to reduced renal perfusion leading to oliguria.

Prerenal AKI is usually reversible if treated appropriately, but any delay in treatment may progress to an intrinsic renal failure (acute tubular necrosis). The resorptive capability of the kidneys is lost in intrinsic renal failure, whereas it is intact in prerenal causes. Due to this, urinalysis can differentiate between the two.

Functional Oliguria and Acute Kidney Injury

Hepatorenal syndrome and drugs such as nonsteroidal anti-inflammatory drugs and angiotensin-converting enzyme inhibitors cause intrarenal hemodynamic functional derangements leading to oliguria. Urinary findings may mimic prerenal renal failure, but may not comply with clinical findings. Improvements in glomerular filtration rate are usually noted after drug discontinuance or management of the liver disease.

Central venous pressure: Generally, a low central venous pressure (CVP) might indicate hypovolemia. However, there are better dynamic indicators of hypovolemia and fluid responsiveness. If cardiac failure is the principal cause of prerenal renal failure, high CVPs are apparent. CVP is a static marker of volume status and is inaccurate.

TABLE 2: Summary of examination of patients with oliguria or renal injury.

S. No.	***Domain***	***Eliciting of signs/symptoms***	***Comments***
1.	Head, face, and neck	• Periorbital edema • Anemia, jaundice—hemolysis, blood loss (hemorrhage and hypotension), reduced production (CKD) • Rash—malar rash, viral exanthems • *Mouth:* – Dry mucus membranes (hypovolemia) – Oral ulcers (sepsis, CTDs) • *Fundus:* Hypertension, systemic infections • Dilated neck veins, elevated or JVP **(Fig. 3)** • Cushingoid features	• This gives a quick clue regarding systemic diseases, and acute vs. chronic renal causes of oliguria/renal failure • Chronic corticosteroid intake can cause cushingoid features
2.	Extremities	• *Fingers and palms:* – *Nail and digits:* Splinter hemorrhages, digital infarcts—IE, vasculitis – Koilonychia—anemia (CKD) • *Hand and legs:* – Skin turgor – *Rash:* TCP, HSP, sepsis, TTP/HUS – Pedal edema—pitting edema – Cellulitis, fasciitis – Cachexia – Asterixis – A-V fistula	• The general examination may through light on the cause of renal failure. Peripheral signs of vasculitis and infective endocarditis are easy to pick up • End-stage renal disease is associated with protein energy wasting (PEW) leading to cachexia
3.	Abdomen, back, and perineum	• *Scars:* Previous history of surgery • *Rash:* Rask in buttocks—HSP • Genital ulcers—systemic diseases and infection • Sacral edema—more relevant in supine patients for dependent edema • Excoriations in perineum and pressure sores—cleaning for frequent loose motions • Phimosis—rare cause in ICU for oliguria • Diaper wet—urine leak and block in Foleys catheter	The perineal and back examination is often missed in ICU patients that may give clues of source of sepsis
	Bedspace and documents	• Vasopressors, NTG infusions • Blood sugars • Urine bag—color quantity, trend of urine per hour	A careful bedspace examination is helpful in getting the trends
4.	Cardiovascular	• JVP, S3, S4, apex beat position • Heart rate, tachy or brady arrhythmias, feeble pulse • Signs of tissue hypoperfusion: Cold peripheries, discoloration, increased CRT, and vasopressor support • Blood pressure: Hypotension/hypertension	Look for signs of low cardiac output and heart failure. The shock of any etiology. These are common cause of prerenal AKI that presents with oliguria. Hypertension may give clues of glomerular pathologies. A quick assessment of the circulatory system by echocardiography is vital. Premorbid blood pressure value may give a clue of the patient's blood pressure range that may be of help in blood pressure targets. (There is some evidence that higher blood pressure targets might improve renal perfusion and function.)
5.	Central nervous system	• Altered sensorium • Muscle weakness and sensory disturbances	• It is common to develop altered sensorium, and many a time it is due to renal failure. • Signs of peripheral neuropathy may raise suspicion of systemic illness and ESRD.

Contd…

Contd…

6.	Respiratory	• Septic focus • *Breathlessness:* B/L crepitation, reduced breath sounds (fluid overload)	• Source of sepsis • Fluid overload—pulmonary edema, pleural effusion
7.	GI	• Vomiting and loose motions—volume depletion • Distended abdomen: Ascites, ileus, increased intra-abdominal pressure	Measuring the loose motions with diaper weight will give a rough idea of the amount of stool volume. Often missed in ICU patients, which adds to unmeasured volume loss. In patients with intra-abdominal pathology, it is important to rule out intra-abdominal hypertension. The common presentation is oliguria and renal failure

(CKD: chronic kidney disease; CRT: capillary refill time; CTD: connective tissue disorder; ESRD: end-stage renal disease; GI: gastrointestinal; HSP: Henoch-Schönlein purpura; HUS: hemolytic uremic syndrome; JVP: jugular venous pulse, IE: infective endocarditis; NTG: nitroglycerin; TCP: thrombocytopenic purpura; TTP: thrombotic thrombocytopenic purpura)

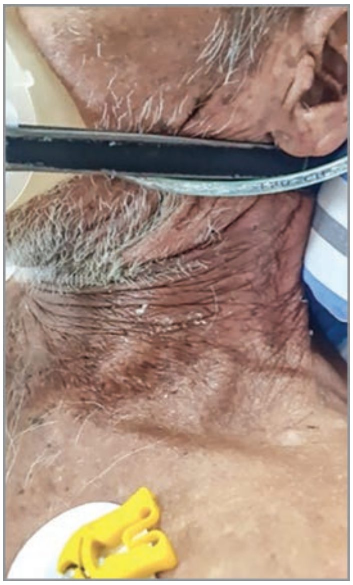


Fig. 3: Dilated neck veins.

Dynamic indices have better predicting ability and is discussed elsewhere.

Fluid challenge: An increase in urine output in response to a carefully administered fluid challenge is both diagnostic and therapeutic in cases of prerenal renal failure. Rapid intravenous administration of 300–500 mL of a balanced crystalloid is the usual initial treatment. Urine output is measured over the subsequent 1–3 hours. A urine volume increase of >50 mL/h is considered a favorable response that warrants continued intravenous infusion. If the urine volume does not increase, the physician should carefully review the results of blood and urine chemistry tests, reassess the patient's fluid status with a POCUS, and repeat the physical examination to determine whether an additional fluid challenge (with or without furosemide) will be worthwhile.

Safety to fill: Oliguria can be associated with fluid overload, congestion, and pulmonary edema leading to hypoxia. Hence, before administering a fluid bolus, it is important to assess the "safety to fill" as vulnerable patients might develop fluid overload and worsening hypoxia. This can be assessed either clinically or combining it with POCUS. Clinical assessment requires the assessment of breathing pattern, oxygenation, and the fluid status. For example, in a patient with cardiogenic shock and oliguria, if the room air oxygen saturation >96% and the respiratory pattern with auscultation is normal, it is generally safe to fill even if the jugular venous pressure (JVP) might be elevated. This might be useful in a resource-limited setting and not in tertiary care centers where supplementation with POCUS has become the standard of practice.

Systemic Examination of the Renal System

The examination of the renal system should be systematic, quick, and comprehensive. The examination can be divided into inspection, palpation, percussion, and auscultation (listen).

Inspection: The examination of the abdomen, especially the lumbar, pelvic, and back is important, as it is concerned with the renal system.

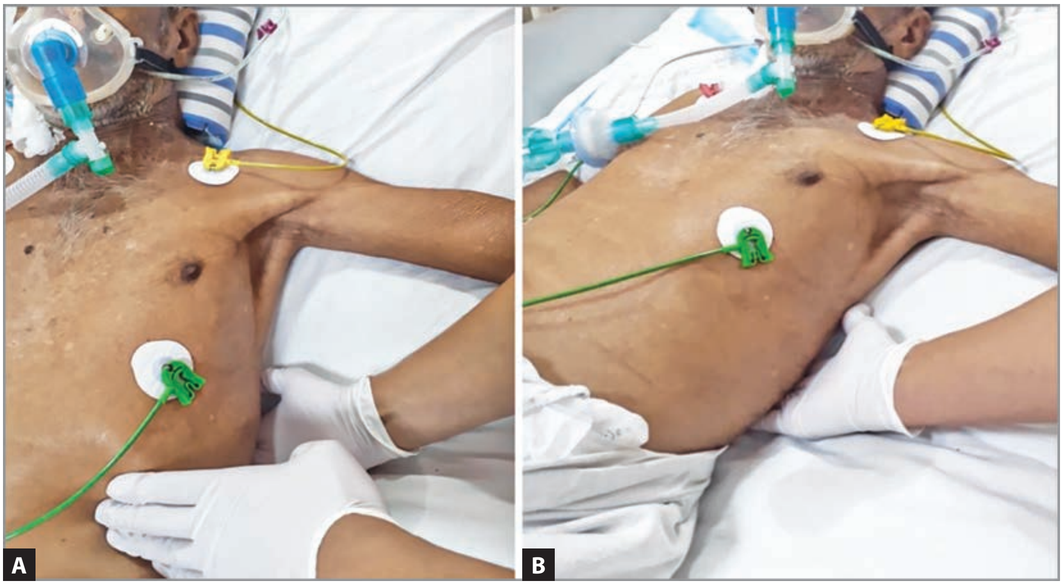
The inspection starts with looking for skin color, scars, sinuses, focal swelling, and examination of the genital and perineum. Discoloration, induration, sinus, and fistula may indicate deep-seated infection. Scars indicate previous renal surgery. Phimosis and urinary catheter kinking are common causes of correctable low output. Swelling—focal distension or swelling at the lumbar region may be an indicator of renomegaly. It is more common in polycystic kidney disease. Umbilicus position—urinary bladder distension, transplant kidney, etc., can alter the position.

Palpation

- *Skin turgor:* It is the measure of the elasticity of the skin. Dehydration leads to loss of elasticity and hence the skin turgor. It is tested by indenting or gently pinching the skin over the sternum. Normally the skin should revert back to its original position. Dehydration leads to slow recovery of the skin to its original state.
- *Kidney size:* Using both hands (bimanual technique) holding the rib cage and the iliac crest, the patient is asked to inhale deeply. The lower pole of the kidneys can be felt during the inspiration. The size, shape, consistency, and mobility can be assessed **(Fig. 4A)**.
- *Presence of focal crepitus:* Palpation over the flank in infections by gas producing organisms leads to crepitus.
- *Warmth:* Warmth might indicate general status of perfusion to all the organs. Localized increase in temperature indicates inflammation or infection like cellulitis.
- *Renal angle tenderness:* Referred pain from cystitis or pyelonephritis leads to tenderness on palpation over the costovertebral angle (Space below the 12th rib joining the vertebra; **Fig. 4B**). Gentle thumping with a fist can elicit this sign **(Fig. 5)**.
- *Urinary bladder size:* Distended bladder can be palpated, which might be due to prostatomegaly or urinary catheter obstruction. POCUS can be used for confirmation
- *Pulsation of aorta:* Aneurysm pulsations are easily transmitted, which might need further investigations.
- *POCUS:* Bedside ultrasound of the kidneys **(Fig. 6)** gives valuable information about the renal parenchymal changes, hydroureteronephrosis, and renal vasculature (Doppler). The renal resistive index (RRI) and renal venous Doppler help to predict the occurrence of AKI and identify a patient with venous congestion.

Percussion

The 12th intercostal space lateral to the spine and the area below (renal angle) it are percussed to assess the kidneys. Normally there is no tenderness. Pyelonephritis and urolithiasis can cause tenderness on percussion.



Figs. 4A and B: Palpating of the kidney. (A) Palpation of the kidney, 2-hand technique. Helps us to know the mobility of kidney and renomegaly; (B) Palpation for renal angle tenderness. Place your hand at renal angle, apply gentle pressure, and look at the patient's facial expression.

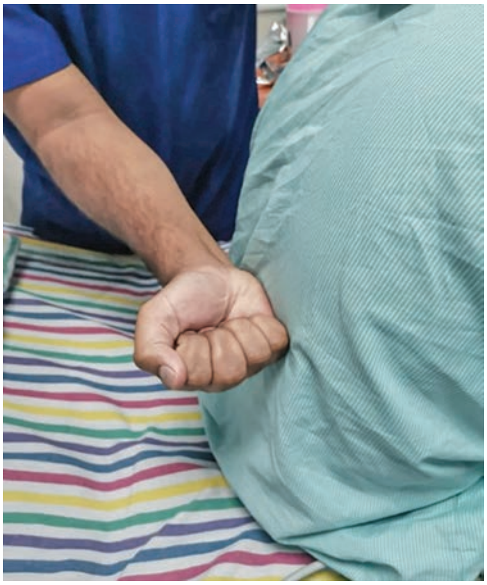


Fig. 5: Gentle fist thumping to elicit renal angle tenderness. The person who is on the front should check the patient's facial expression and enquire about pain.

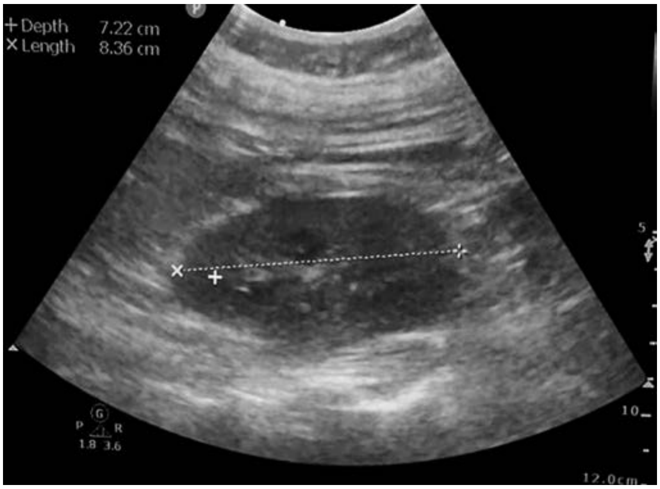


Fig. 6: Ultrasound image showing normal kidneys.

Auscultation: Auscultation just lateral to the umbilicus for renal artery bruit and over the midline for abdominal aorta bruit could reveal arterial narrowing secondary to atherosclerosis, arterial stenosis, or occlusion. This will require further ultrasound Doppler examination or a CT scan for confirmation.

A brief approach to oliguria is described in **Flowchart 1**.

Other System Examination

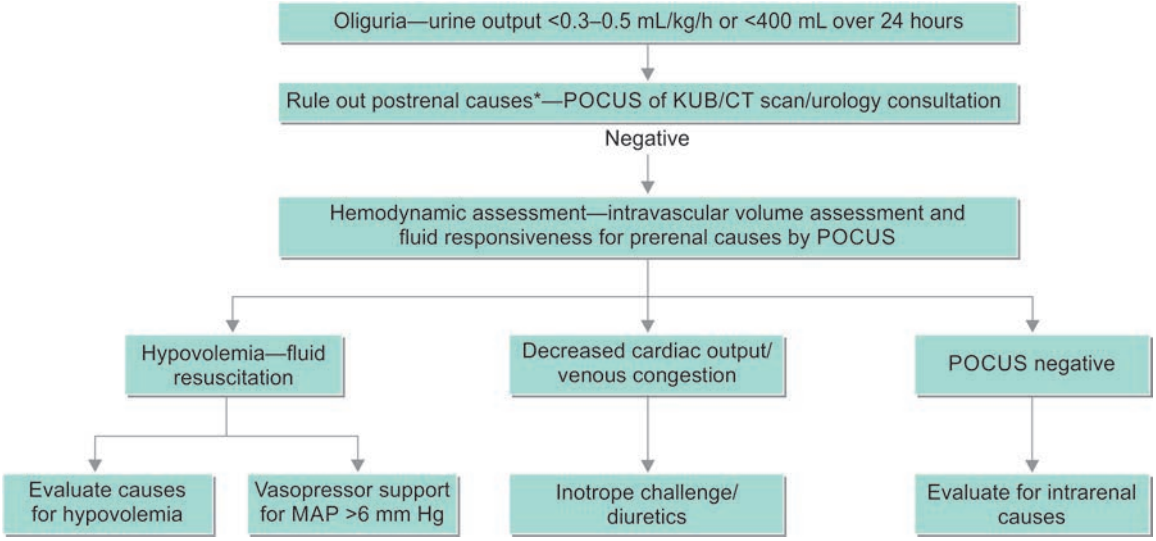
The examination of other systems is very important in finding the cause of oliguria, organ compromise due to renal injury, and to identify distant complications. The examination should be focused and quick. **Table 2** provides the details.

- *Respiratory system:* Oliguria often leads to fluid accumulation and could lead to pulmonary edema resulting in hypoxia. On inspection, the patient might appear restless or anxious, there might be tachypnea associated with sweating. Palpation and percussion might be unremarkable. Auscultation can reveal crepitations due to fluid overload. POCUS is invaluable in such situations.
- *Cardiovascular system:* Examination of the cardiovascular system might reveal either the cause or the effects secondary to oliguria. A low JVP due to hypovolemia might reveal the cause, and on the other hand, a high JVP might indicate a volume overload status secondary to oliguria or due to cardiac failure. Palpation and percussion might be unremarkable. Auscultation might reveal abnormal S3 or S4 due to heart failure.

Investigations

Evaluation of oliguria should include renal function tests and serum electrolytes. A urine routine microscopy can give a wealth of information. A careful look at the patient chart will give ample details on the cause of oliguria and its progress. An extended examination to look at the chart for further investigations such as autoimmune profile, renal ultrasound and Doppler, cystoscopy, and CT abdomen might be needed. The evaluation needs to be aggressive since progression to acute renal failure can result in worse outcomes.

Flowchart 1: Approach to oliguria.


Oliguria—urine output <0.3–0.5 mL/kg/h or <400 mL over 24 hours
Rule out postrenal causes*—POCUS of KUB/CT scan/urology consultation
Negative
Hemodynamic assessment—intravascular volume assessment and fluid responsiveness for prerenal causes by POCUS
Hypovolemia—fluid resuscitation
Decreased cardiac output/ venous congestion
POCUS negative
Evaluate causes for hypovolemia
Vasopressor support for MAP >6 mm Hg
Inotrope challenge/ diuretics
Evaluate for intrarenal causes

(CT: computed tomography; MAP: mean arterial pressure; POCUS: point-of-care ultrasound; KUB: kidney, ureter, and bladder)
*Foley catheter obstruction, ureteric calculi/hydroureteronephrosis, prostatomegaly

SUMMARY

Oliguria can be a symptom or a sign, which indicates low urine output. Assessment of renal perfusion and patency of the urogenital system is critical to establish the cause. Oliguria is reversible when detected and acted upon early. History, clinical examination and ultrasound assessment must be combined to treat oliguria and POCUS can also be used to monitor its progress after intervention. Progression to acute kidney injury leads to worse outcomes.

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Chapter

36

How will I Approach/Examine a Patient with Suspected Vascular Insufficiency/Peripheral Vascular Disease?

Gautham M Raju

ABSTRACT

Peripheral vascular disease (PVD) is a relatively common medical condition that affects the vasculature and blood flow across many parts of the body. Peripheral arterial disease (PAD) and chronic venous insufficiency (CVI) are the most common presentations of PVD and result in significant morbidity. Vascular insufficiency can also occur in the distribution of the aorta and central vessels such as mesenteric and renal vasculature. In critically-ill patients, vascular insufficiency can occur frequently and can cause significant debilitation during the illness or recovery process. A systematic approach to history, general and focused systemic examination with regular reassessment of vascular status can help avert major catastrophes and improve patient outcomes.

INTRODUCTION

This chapter focuses on an approach to a patient with vascular insufficiency and/or peripheral vascular disease (PVD) in critically ill.

In general, the most common causes of vascular insufficiency are peripheral arterial disease (PAD) and chronic venous insufficiency (CVI). "PAD is chronic progressive atherosclerotic disease leading to partial or total peripheral vascular occlusion." Typical areas PAD affects are abdominal aorta, iliac arteries, arteries of the lower limb, and occasionally arteries of the upper limb. PAD presence can range from being asymptomatic to significant occlusive disease [critical limb ischemia (CLI)] including ulceration, claudication, ischemia limb loss (especially in extremities), and is associated with high risk fatal and nonfatal cardiovascular adverse events. While PVD in the general population adds a significant burden to morbidity, the development of vascular insufficiency acutely at presentation or occurring during the intensive care unit (ICU) stay as a secondary consequence adds to both morbidity and mortality in the ICU.

Moreover, in critical illness, the complex physiological and pathological states can bring about a dynamic shift in blood flow across arterial and venous systems over relatively short periods, can result in end-organ dysfunction, and loss of limb or life.

In critically-ill patients it can occur at various locations. Excluding mainstream vascular emergencies like stroke or acute coronary syndrome, they can be of clinical importance in extremities and mesenteric arterial or venous systems; and can result in considerable morbidity and mortality in critically-ill patients. Many of the issues can arise during their stay in the ICU, the keen appraisal of the onset and/or progression being very valuable in averting the progression of the problem.

CASE SCENARIO

A 58-year-old lady with a history of systemic lupus erythematosus (SLE) with lupus nephritis on regular dialysis was admitted to the hospital for urosepsis [left-sided pyelonephritis with significant hydroureteronephrosis (HUN)] with septic shock. The patient also had a history of diabetes mellitus and hypertension. The patient was in an immunosuppressed state with long-term steroids for underlying SLE before hospital admission.

During this period the patient underwent treatment in the ICU where patient needed multiple vasopressors for several days. The patient was noted to have bluish discoloration of bilateral forefeet (left > right). She was also noted to have discoloration of her feet with mottling in the skin and signs of tissue hypoperfusion and poor capillary refill time (CRT). Even though over time the aforementioned signs improved with the weaning of vasopressors, patient went on to develop dry gangrene in the first and second toe in the right foot and ulceration in other toes in both feet.

She was hospitalized for a total duration of about 2 months. During this time, she underwent source control with left-sided double J (DJ) stenting and drainage of purulent material. She was on antibiotics for the same and successfully recovered. However, with the demarcation of the ischemic injury and limited tissue loss, decision to consider amputation of the affected foot was delayed till she recovered from the acute illness.

Postrecovery from urinary tract infection, on clinical evaluation the patient was noted to have absent distal pulse in the right lower limb. Patient underwent angiography before surgery was considered. Angiography showed serial stenosis in distal superficial femoral artery and severe high-grade stenosis in the mid-popliteal artery with poor flow distal to affected area. Patient underwent percutaneous transluminal angioplasty of the lesions and subsequently underwent limited forefoot amputation which healed uneventfully.

HISTORY AND SYMPTOMATOLOGY

History

While approaching a patient who is experiencing symptoms related to vascular function, it is important to approach history taking in lines of the risk factors associated with the following factors:

- *Peripheral vascular disease in general:* Risk factors that largely promote or worsen atherosclerosis,
- Vascular issues in critically ill.

Box 1 enumerates common risk factors associated with peripheral vascular disease in general.

It is prudent to keep the "6 P's" of acute limb ischemia in mind as part of history taking.

1. Pain
2. Pallor
3. Paresthesia
4. Paralysis
5. Pulselessness
6. Poikilothermia

Box 2 enumerates common risk factors associated with development of impaired peripheral perfusion in the critically ill.

Table 1 gives an integrated overview of clinicopathological concepts regarding the history and symptomatology of peripheral vascular insufficiency.

BOX 1: Risk factors associated with peripheral vascular disease.

- Diabetes mellitus
- Hypertension
- Age
- Family history of cardiovascular disease
- Tobacco use
- Hyperlipidemia, hyperhomocysteinemia, and hyperfibrinogenemia
- Sedentary lifestyle

BOX 2: Risk factors associated with vascular issues in critically ill.

- Systemic inflammatory response syndrome, sepsis
- Thrombotic/embolic phenomena and hypercoagulable states
- Circulatory failure and microvascular disturbances
- Use of vascular indwelling devices, e.g., arterial line
- Use of vasoactive agents, e.g., noradrenaline, adrenaline, and vasopressin
- Chronic stenotic lesions, e.g., atherosclerosis

EXAMINATION OF PATIENTS

Examination of a patient with vascular disturbances should proceed systematically keeping certain things in mind as follows:

- In the ICU, vascular insufficiency can occur in two ways:
    1. Preexisting vascular disease or systemic disease with vascular involvement, presenting as an exacerbation.
    2. No obvious preexisting disease but arising out of relative insufficiency due to pathologic processes (e.g., thrombotic, embolic phenomena, and inflammatory processes) or iatrogenic processes (use of vasoactive agents, use of intravascular devices such as arterial catheter, venous catheter).
- *Pathophysiological implications:* The vascular insufficiency can occur due to disturbances in the blood flow in the following systems:
    - *Arterial system:* Starting from ascending aorta to peripheral arteries.
    - *Venous system:* Starting from peripheral veins to the venae cavae.
    - Secondary occlusion of arterial system, venous system, or both; due to interstitial edema or pathological conditions like compartment syndrome in predisposed areas of the body.

In critically-ill patients, it can occur at various locations. Excluding mainstream vascular emergencies

TABLE 1: History and symptomatology—peripheral vascular insufficiency.

S. No.	***Domain***	***Relevant history to note***	***Comments***
1.	Presenting illness	• *Pain*—claudication pain is classical of peripheral arterial insufficiency. Increase with activity and reduces when the activity is stopped • *Pain at rest*—due to nerves damage, start from tip of fingers or toes. Also, may indicate critical limb ischemia *Discoloration:* • Pale—more with acute ischemia. Congested in case of venous occlusion • Cyanosed—late phase of arterial or venous occlusions • Gangrene—late stage, commonly dry gangrene, when chronic	Application of warmth will aggravate the pain in arterial insufficiency. Raynaud's phenomenon—application of cold object: digits turn pale, the blue and back to normal color when temperature is back to normal
2.	Previous history and medication history	*Known PAD:* • Raynaud's, Berger's disease • Atherosclerosis, hypercholesterolemia • Previous episodes of vascular insufficiency and treatment received *Medications:* • Drug overdose/poisoning—ergot derivative • Recent administration of noradrenaline, vasopressin	Patient who are known case of PAD are at risk of developing limb/mesenteric ischemia. Rare cause of digital ischemia are adverse events due to drugs in general population. In critically-ill patients severe shock in high-risk individual and use of vasopressors are important history to note
3.	• Temporal events • The details help us to find the casual association to the event	*Current/recent illness:* • High dose vasopressor use • Thromboembolic phenomenon (infective endocarditis, hypercoagulable states) • Polytrauma • Catastrophic APLA • Sepsis and DIC • Bed ridden, long hospitalization (at risk of DVT and venous insufficiency) *Recent surgery or instrumentation:* • Major surgery (aortic dissection or aneurysm surgery are prone for distal ischemia) • Arterial line (femoral or radial cannulations—for arterial blood pressure monitoring or for procedures like vascular interventions for myocardial infarction, stroke, etc.) • Femoral arterial cannulation especially VA ECMO or IABP or percutaneous ventricular assist devices	• Many times, temporal events give clue on the possible etiologies of digital gangrene/discoloration • Mesenteric ischemia is often challenging to diagnose, needs a high suspicion in high-risk patients
4.	• Comorbid conditions and personal habits • Confounders for assessment, aggravate the existing severity of disease and help to choose perfusion targets	• *Atherosclerosis and complications:* CAD, stroke • *CAD, DCM/HF, valvular heart disease, circulatory failure:* Confound the assessment, low blood flow states may further reduce the distal flow of the obstructed vessel • *Autoimmune, CTDs—peripheral vascular diseases:* Claudication pain, Raynaud's phenomenon—may confound assessment or different BP values in both sides • *Malignancies:* Watch for chemotherapy related adverse effects, prone for venous thrombosis • *Hematological diseases:* Sickle cell disease, thrombophilia, polycythemia, and myeloproliferative disorders • *Smoking and drug addiction*—increased risk of vascular complications	Suboptimal control of comorbid conditions and systemic disease increases the potential risk of organ dysfunction. Information regarding systemic diseases will help us to do focused evaluation

(APLA: antiphospholipid antibodies; BP: blood pressure; CAD: coronary artery disease; CTDs: connective tissue diseases; DCM: dilated cardiomyopathy; DIC: disseminated intravascular coagulation; DVT: deep vein thrombosis; HF: heart failure; IABP: intra-aortic balloon pump; PAD: peripheral arterial disease; VA/ECMO: venoarterial extracorporeal membrane oxygenation)

like stroke or acute coronary syndrome, they can be of clinical importance in extremities and mesenteric arterial or venous systems; and can result in considerable morbidity and mortality in critically-ill patients. Many of the issues can arise during their stay in the ICU, the keen appraisal of the onset and/or progression with respect to degree and severity, being very valuable in averting further advancement of the problem.

General Physical Examination

In the ICU, it should be kept in mind that general or systemic examination is performed in light of clinical signs and symptoms, history, and underlying risk factors in the view to ascertain the extent of the damage due to the vascular disturbances and to help plan appropriate management **(Table 2)**.

Focused Examination

- Vital parameters [blood pressure, pulse rate, respiratory rate, oxygen saturation (SpO_2), Glasgow Coma Scale], presence of invasive lines and their functional status; all at presentation as standard assessment in any patient coming into the ICU.
- Keeping above-mentioned case scenario, the patient is suffering from SLE, which is multisystem disorder, also look for the following signs:
  - Signs of mucocutaneous inflammation in predisposed areas
  - Peripheral areas for signs of preexisting or ongoing vasculitic phenomena (e.g., Raynaud's phenomena)
  - Joints and peripheral edema
  - Pigmentation, ulceration, cellulitis

TABLE 2: Initial empirical schema in approach to vascular insufficiency.

S. No.	*Parameter being monitored*	*Relevant signs/features due to vascular insufficiency*	*Comments*
1.	Tissue perfusion and clinical assessment in circulatory/vascular insufficiency	Central nervous system changes	Altered sensorium, delirium confusion
		Respiratory system changes	Deep breathing, tachypnea (due to acidosis)
		Renal system changes	Oligoanuria
		Gastrointestinal tract changes	Abdominal pain, distension, pulsatile mass (aortic aneurysm), bowel dysfunction, diarrhea/constipation, bloody stools (mesenteric ischemia/aortoenteric fistula)
		Extremity changes	Changes in skin color, mottling, long capillary refill time, compartment syndrome (if undetected or untreated can lead to nerve and muscle ischemia), stigmata of vasculitis (red spots/lumps/ulcers)
		Cardiovascular changes (given further)	
2.	Laboratory changes	Blood lactates	• Elevated due to anerobic metabolism due to lack of effective perfusion and oxygen delivery • Significant and progressive elevations occur in mesenteric ischemia along with hemoconcentrations (due to reduced intravascular volume) and neutrophilic leukocytosis
3.	Cardiovascular changes	• Basic noninvasive monitoring • Echocardiography • Cardiac output monitoring devices	• Changes in BP/MAP/pulse pressure • Assessment of stroke volume, cardiac output, valvular diseases [damage/clots/vegetations (infective endocarditis)] • Both invasive and noninvasive cardiac output monitoring devices can be used to measure cardiac output and circulatory function to help wean vasoactive agents (excessive or prolonged use of which can itself precipitate end-organ perfusion defects)

(BP: blood pressure; MAP: mean arterial pressure)

Note: It is important to note that in critically-ill patients, involvement of multiple organ systems is quite frequent and examination proceeds accordingly.

Systemic Examination

Systemic examination of a patient in intensive care needs to proceed keeping in mind the organ system involved (intestine, visceral organs, mesenteric vasculature, etc.).

Approach to a patient with PVD is discussed keeping the above case scenario in mind.

Conventional Approach

Table 3 illustrates conventional approach to examination which includes inspection, palpation, auscultation, and ancillary tests.

Assessment of Peripheral Perfusion in ICU

Peripheral perfusion is an important function of the cardiovascular system whose disturbances are quite frequent in the ICU. Systematic and regular measurements can help effective management of hemodynamics of the critically-ill patients and avert end-organ catastrophes (with consequent loss of limb or life).

It can be followed with assessing the following at bedside:

- Systematic skin temperature assessment of the extremities
- Mottling of skin
- Capillary refill time

Skin Temperature Examination in ICU

It can reflect the circulating blood volume within the superficial blood vessels of the limbs.

As per the currently available studies, skin temperature is systematically measured by subjective means (palpation from distal to proximal end of the extremity) and by objective means using skin temperature measuring devices.

Even though studies do not show conclusive evidence for using skin temperature as a definitive indicator of measure of hypoperfusion, it can be valuable for real time use as a noninvasive measure to ascertain hypoperfusion.

Mottling of Skin

Lacy net-like purplish discoloration on the skin due to sluggish blood flow in the dilated superficial postcapillary venules.

Capillary Refill Time

- *What is capillary refill time?*
  Capillary refill time is a quick, noninvasive, fairly reliable method of detecting changes in blood flow that can lead to shock. Normal values being about 2 seconds. Slightly longer in women, elderly, and cold ambient temperature.
- *How to perform and interpret?*
  The healthcare provider applies a firm pressure on the skin of the plantar surface of the great toe for 5 seconds in noncritically-ill patients and 15 seconds in the critically-ill patient, and then it is timed how long it takes for the skin to come back to normal in seconds. Measurement of >5 seconds (5s) is regarded as abnormal. Signs of hypoperfusion are illustrated in **Figure 1**.

Note: When there is reduction in cardiac output or regional loss of effective blood circulation during the course of critical illness, the body responds by reduction in the cutaneous blood flow which may produce cooler limbs, mottling, and longer capillary times; which each by themselves or in combination with the others can

TABLE 3: Conventional approach to examination.

Inspection	*Palpation*	*Auscultation*	*Ancillary test*
Color, texture changes in the skin of extremities	Skin temperature difference in affected limb (temperature lower than normal limb)	Limb bruit present (when auscultated above affected section of the vessel)	Capillary refill time >5 seconds
Ulcerated/atrophic/ edematous skin	Absent femoral pulse absent		Venous filling time >20 seconds
Absent hairs on extremities	Absent posterior tibial and/or dorsalis pedis pulse		Vascular Doppler

Source: Adapted from McGee. Peripheral vascular disease. Evidence-Based Physical Diagnosis; 2018.pp.461-8.e2.

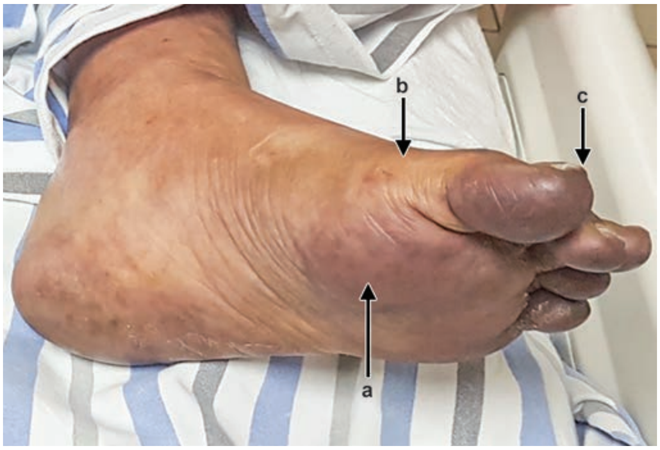


Fig. 1: Feet of patient with newly detected vascular insufficiency showing signs of impaired peripheral perfusion. (a) Altered color (compared with surrounding area) suggestive of initial appearance of poor tissue perfusion; (b) Mottling (lacy net-like discoloration); and (c) Peripheral cyanosis (vascular insufficiency getting well established). These signs should prompt the clinician to examine and evaluate further.

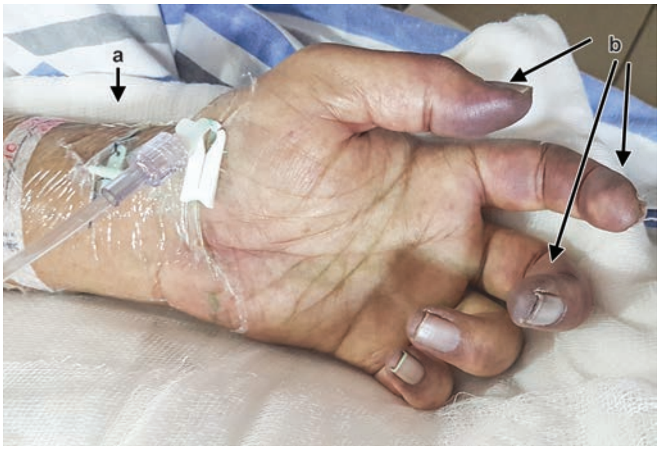


Fig. 2: Hand of the same patient illustrating features of acute limb ischemia. (a) Indwelling arterial catheter; and (b) Peripheral cyanosis in the fingers distal to the distribution of radial artery suggestive of impaired blood flow.

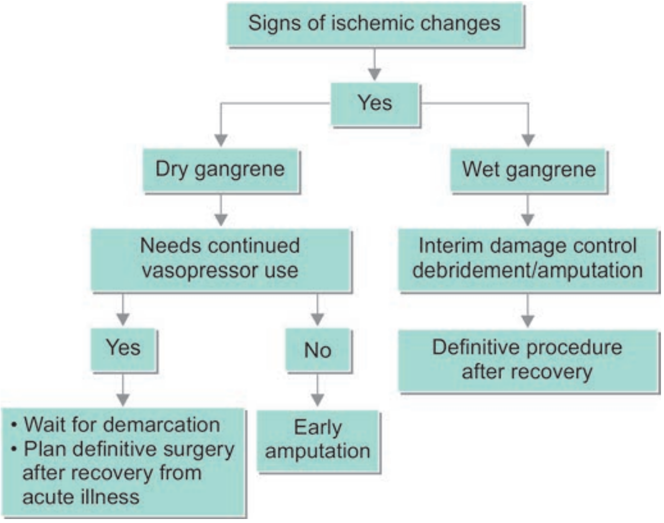
predict the following: Low cardiac output, multiorgan dysfunction, major postoperative complications after intra-abdominal surgery, mortality, or morbidity in cases of limb ischemia.

Adjuvants to Examination

- *Doppler ultrasound flow study:* Ultrasonic waves are used to ascertain presence and nature of blood flow in affected vessels which may help identify and quantify the degree of vascular flow disturbance. Normal: Triphasic flow; abnormal: Monophasic or absent flow.
- *Angiography:* Contrast imaging study of the vascular systems under investigation. It can give valuable information of structure and patency of the entire length of the blood vessel. Helps quantification of the disturbance and even plan appropriate management.
- *Magnetic resonance angiography:* Noninvasive imaging study similar to angiography.
- *Photoplethysmography (PPG):* Another noninvasive technique where a small blood pressure cuff and a PPG sensor (emits and measures infrared light on surface of skin) is used to measure blood flow in the extremity. Gives readings in form of waveforms which can be used for interpretation.

Note: When examining critically-ill patients who require urgent evaluation and further management, these other adjuvants may quickly help to rule out differentials during the examination. Repeat the test/assessment at every shift or everyday as part of FASTHUG checklist to ensure crucial findings are reliably identified.

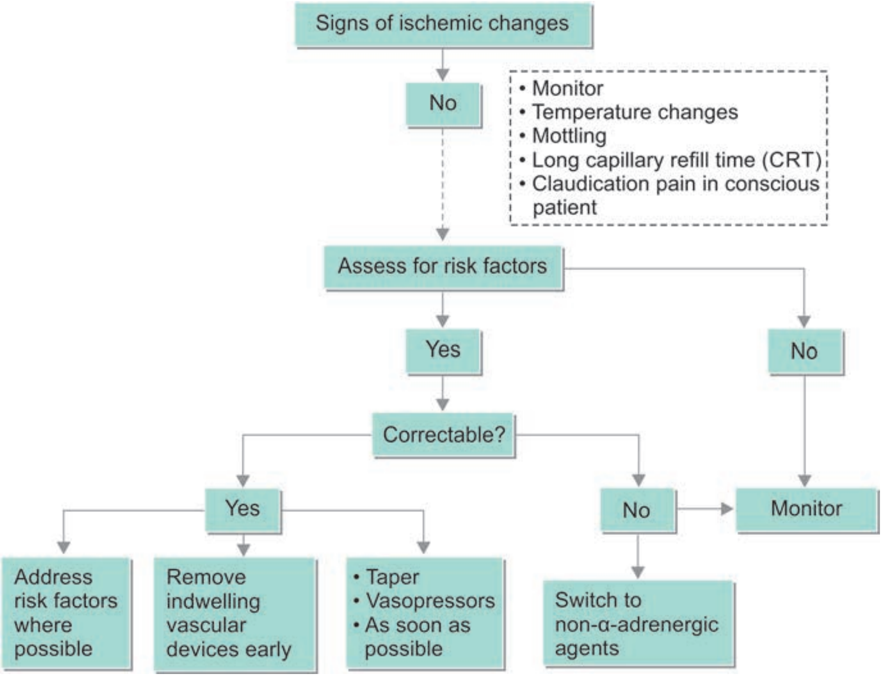
Flowchart 1: Approach to patient "with" signs of ischemia in extremities.


Signs of ischemic changes
Yes
Dry gangrene
Wet gangrene
Needs continued vasopressor use
Interim damage control debridement/amputation
Yes
No
Definitive procedure after recovery
• Wait for demarcation
• Plan definitive surgery after recovery from acute illness
Early amputation

Keeping the **Figure 2** and case scenario in mind, approach to patient with vascular insufficiency in the ICU needs a systematic approach. A schematic approach is given in the flowcharts.

Flowcharts 1 and 2 shows the schematic approach to examine a patient with regards to presence **(Flowchart 1)** or absence **(Flowchart 2)** of ischemic changes in extremities.

Flowchart 2: Approach to patient "without" signs of ischemia in extremities.


Signs of ischemic changes
No
• Monitor
• Temperature changes
• Mottling
• Long capillary refill time (CRT)
• Claudication pain in conscious patient
Assess for risk factors
Yes
No
Correctable?
Yes
No
Monitor
Address risk factors where possible
Remove indwelling vascular devices early
• Taper
• Vasopressors
• As soon as possible
Switch to non-α-adrenergic agents

Source: Adapted from Newbury A, Harper KD, Trionfo A, Ramsey FV, Thoder JJ. Why not life and limb? Vasopressor use in intensive care unit patients the cause of acute limb ischemia. Hand (NY). 2020;15(2):177-84.

MESENTERIC ISCHEMIA

Mesenteric ischemia is one of the most catastrophic forms of vascular insufficiency with very high morbidity and mortality.

There are mainly two types of mesenteric ischemia:

1. *Occlusive mesenteric ischemia:*
   i. Thrombotic/embolic
   ii. Arterial/venous
2. *Nonocclusive mesenteric ischemia (NOMI):* Profound hypotension

   Presence of excessive exogenous or endogenous vasoactive substances usually present late as symptoms occur only after large portion of vascularity of intestines are compromised.

Clinical Features

Severe abdominal pain, vomiting, and diarrhea (with or without blood) in initial stages but later can present with signs of ischemia and peritonitis (may indicate irreversible ischemia). Clinical features may be associated with lactic acidosis (which is usually a sign of anerobic metabolism due to impaired regional perfusion), hemoconcentration, and neutrophilic leukocytosis. In view of gastrointestinal dysfunction being quite frequent in the ICU, high index of suspicion and emergent computed tomography (CT) angiography [biphasic (early arterial phase and delayed venous phase) contrast CT protocol] is necessary for timely management.

SUMMARY

- Peripheral vascular disease involves both arterial and venous insufficiency which are usually caused by chronic atherosclerotic disease and valvular dysfunction, respectively.
- In the ICU, patients can suffer vascular insufficiency due to vascular system being acutely impacted (commonly secondary to need for high dose or duration of vasoactive agents) or worsening in the background of prior predisposing factors (atherosclerosis, inflammatory diseases affecting vascular systems focally, e.g., vasculitides, or systemic involvement, e.g., SLE, etc.)
- History of prior conditions may help preclude and manage the disturbance in blood flow.
- Systematic examination of the different areas of the body, especially the peripheries, with respect to assessment of palpable pulses in crucial arteries is

important. In patients with significant circulatory failure, assessment of skin temperature and capillary refill can be valuable indicators of signs of perfusion. Lactic acidosis and gastrointestinal dysfunction can indicate mesenteric vascular insufficiency.

- History and examination can be efficiently supported by adjuvant radiological assessment. Doppler studies are especially useful at bedside and can help decide and utilize advanced imaging such as computed tomographic angiography (CTA) and magnetic resonance angiography for further characterizing or planning management.
- It is noteworthy that vascular systems face significant brunt because of the various systemic upheavals that occur during the course of critical illness. Early recognition, assessment, quantification, and rectification of the disturbances is crucial to prevention and/or address the catastrophic consequences of vascular failure.

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Chapter

37

How will I Approach/ Examine a Patient who is a Potential Organ Donor?

Rahul Anil Pandit

ABSTRACT

The chapter focuses on how to approach and examine a patient who is potentially brain dead. While all efforts in intensive care unit (ICU) are directed toward saving life, there may be some clinical scenarios where despite best of the efforts there may be no brainstem function elicited on clinical examination, or there may be a rapidly declining neurological condition, which has a potential to become brain dead.

The chapter focuses on such patients, what are the clinical conditions which could progress to brain dead, clinical mimics which can be confounders, preconditions, and then how to diagnose brain dead. Most important aspect is to maintain a potential brain-dead patient, as they may become an organ donor, and hence maintaining a normal physiology is of paramount importance.

INTRODUCTION

The critical care physician plays an essential role in identifying and assessing potential organ donors. Identifying potentially brain-dead patients is crucial, as diagnosing brain death and managing potential organ donors is of utmost importance. Physicians should be familiar with evaluating patients for brain death and should also understand local laws, regulations, and ethical guidelines related to organ donation, considering the patient's autonomy and the wishes of the patient and their family.

The evaluation should begin by gathering relevant medical history and supporting evidence pointing toward permanent brain damage. This includes the patient's medical history, chronic illnesses, infections, surgeries, and lifestyle factors. It is important to identify any contraindications to organ donation, such as active malignancy, human immunodeficiency virus (HIV), or severe sepsis. A comprehensive physical examination, including examination of the central nervous system and systemic evaluation, is essential. Additionally, reviewing brain imaging and relevant lab parameters, such as serum sodium and urea levels, is important as these factors may confound the examinations. Every critical care physician should possess the necessary skills to effectively examine patients in a coma and potentially brain-dead patients. Systemic examination and documentation of findings are crucial in this process. This chapter provides a systematic approach for evaluating patients who may be potential organ donors.

CASE SCENARIO

A 22-year-old male suffered road traffic accident; emergency medical services noticed cardiac arrest while transporting to hospital; and the patient received cardiopulmonary resuscitation (CPR) during the transport. Patient reviewed.

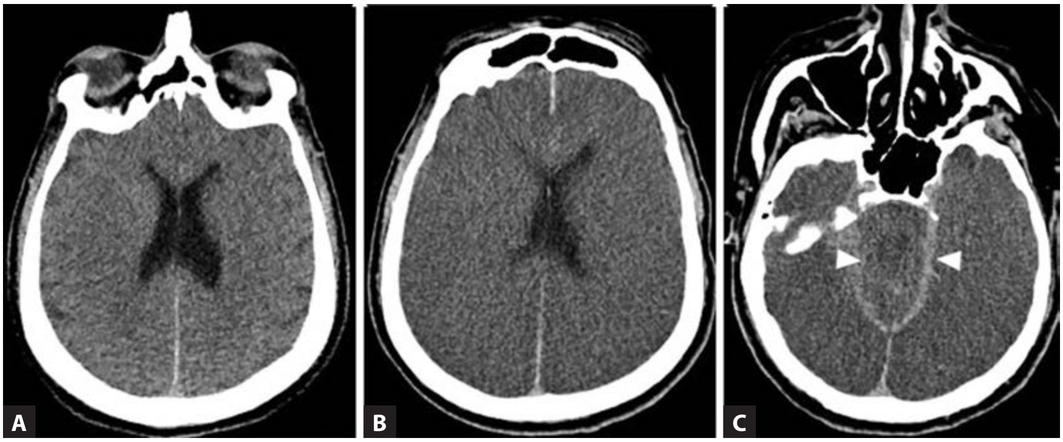
On admission to emergency room (ER), the patient was unconscious; Glasgow Coma Scale (GCS) was E1 M3 Vt; pupils dilated fixed; not reacting to light; blood pressure (BP) 90/60 mm Hg; and on mechanical ventilation.

Patient's computed tomography (CT) imaging study was suggestive of catastrophic brain injury ***(Figures 1A to C)****.*

EXAMINATION OF PATIENT

History

A detailed clinical history should be noted, right from information available at scene (if any), what was the nature of accident, high velocity/motor bike, or if helmet was used. These questions often help us to understand the gravity of the impact due to accident.



Figs. 1A to C: Computed tomography (CT) images of a patient who presented to hospital following cardiac arrest with anoxic brain injury. Initial noncontrast CT image obtained <2 hours following initial arrest. (A) Early loss of gray–white matter differentiation of the cerebral cortex. Follow-up study 36 hours later; (B and C) Progression of loss of gray–white matter differentiation including the visualized brainstem with increased cerebral edema, sulcal and ventricular effacement, and effacement of the basilar cisterns.

First responders and ambulance notes need to be looked in great detail; perhaps a direct hand over from ambulance paramedic or doctor is important. The duration of arrest, presenting rhythm, and possible cause of arrest help in understanding the clinical condition and prognostication.

Symptomatology

Never rush into clinical examination, always observe at the bed side. A lot of information can be gathered. Start with monitor, looking at electrocardiogram (ECG), rate, rhythm, BP, temperature, and saturation. Often potential brain dead patients are hypotensive and need vasopressors. Also, it is common for them to be hypothermic as body's ability to maintain temperature is lost. A very large dilute urine output every hour points toward diabetes insipidus.

General Physical Examination

- Start from neurological assessment of GCS.
- Ask for any sedatives or muscle relaxant used. They often confound clinical examination.
- Check for heart rate, rhythm, BP, and temperature.
- Ventilatory settings, any trigger noted by nurse on examination is important. Absence of trigger points toward brain death.
- Look for injuries, and confirm injuries on X-ray or scans.

Systemic Examination

A thorough clinical examination of the neurological system is important.

It is divided into three clinical principles, which are prerequisites for brain death:

1. Documentation of coma
2. Absence of brainstem reflexes
3. Presence of apnea on apnea testing.

All of the above are true in absence of any confounders or clinical mimics.

First step is to confirm if clinical condition supports diagnosis of brain death.

It is imperative to confirm if the patient has a clinical condition supported by history and CT scan or magnetic resonance imaging (MRI), which can progress to brain death. If yes, please progress ahead, if no then consider other clinical diagnosis.

Brain death is commonly caused by but not limited due to the following:

- Intracranial hemorrhage or subarachnoid hemorrhage
- Ischemic brain injury
- Head injury
- Cerebral anoxia/ischemic injury due to any cause
- Primary cerebral tumor

Confounders that need to be ruled out:

- *Presence of sedatives or muscle relaxants:* Wait for at least 5 half-life of the drugs to be eliminated before testing (in patients with normal renal and liver function).
  If in doubt, we may wait longer, or consider dialysis.
- *Hypothermia:* Always keep patient at a warm temperature of 36°C.

- No abnormal endocrine function, e.g., normal blood glucose
- No toxins, poison, or history of intoxication
- No metabolic abnormality—check sodium, potassium, and acid-base balance

Clinical Mimics of Brain Stem Death

- Locked-in syndrome
- Metabolic coma
- Endocrine coma
- Nerve toxins such as snake bite and muscle relaxants.

Once the above has been confirmed, then clinical systemic examination for brain death should be proceeded.

Systemic Neurological Examination: Clinical Testing for Brain Stem Death

The three clinical principles will cover the brainstem examination in detail. Brainstem death is a completely clinical diagnosis.

1. *Documentation of coma:* This clinical test is done to demonstrate absence of any response to deep painful stimulus in the cranial nerve distribution area **(Table 1)**.
   How to test:
   - Patient's limbs are well exposed and a firm supraorbital pressure is given below each eyebrow at the medical canthus of eyeball. Here the ophthalmic branch of the trigeminal nerve is located in a groove. Both the efferent and afferent responses are from the cranial nerve. No response to pain in form of *no* grimace on face or *no* limb movements shall confirm coma.
   - Other areas where the cranial nerve can be stimulated for pain response are jaw lift or trapezium squeeze at the nape of neck. The trigeminal and spinal accessory nerves are simulated respectively.
   - We often start with providing a graded pain stimulus at the sternum. Should a sternal rub be done during the "NO response to pain stage"? This often stimulates spinal dermatomes and inflicts a spinal response, which would be confounding in clinical examination.
2. *Documentation of absence of cranial nerve function:* Rostral to caudal cranial nerve examination should be performed to demonstrate absence of cranial nerve function.
   - Absence of pupillary reflex response to light
   - Absence of corneal reflexes
   - Absence of vestibulo-ocular reflexes
   - Oculocephalic reflex
   - Absence of gag
   - Absence of cough reflexes
     i. *Absence of pupillary reflex response to light:* Shine a bright narrow beam of light into each eye. No eye drops to dilate the pupils should have been used in the 4 hours previously. The pupils need *not* be fully dilated, could be mid-level 3 mm or more in size in the brainstem dead patients, but there should be no pupillary response to light. Absence of direct or consensual reflex should be demonstrated.
     ii. *Absence of corneal reflex:* A wisp of cotton should be teased off the tip of a moist cotton swab and should be used to lightly stroke the cornea without damaging it. Bilateral absence of corneal reflex should be demonstrated.
     iii. *Absence of vestibulo-ocular reflexes:* Examine both ears to ensure they are wax free and ear drum is intact.

     Also called the cold caloric test, this involves instilling a large volume (50 mL) of ice-cold water into the external auditory canal. This stimulates the lateral semicircular canal and causes tonic movement of the eyes to the same side (the fast component if nystagmus was present would have been to the opposite side).

TABLE 1: Clinical testing for cranial nerves to diagnose absence of brainstem reflexes.

Brainstem reflexes	***Sensory cranial nerve***	***Motor cranial nerve***
Absence of pupillary response to light	II	III (parasympathetic nerve fibers)
Absence of corneal reflexes	V	VII
Absence of cranial nerve response to pain	V	VII (and limb motor responses)
Absence of vestibulo-ocular reflexes	VIII	III and VI
Absence of gag and cough reflexes	IX, X	X

To maximize the stimulus, flex the head to 30°—this will make the semicircular canal vertical. If there is no movement of the eyes (observe for at least 30 seconds after irrigation), the reflex is absent.

iv. *Absence of oculocephalic reflex:* Should only be done in patients who have no documented cervical spine injury.

Holding the eyes open, the patient's head is rapidly turned to one side. If the reflex is present, the eyes will turn to the opposite side, giving the impression that the eyeballs have not moved. If the reflex is absent, the eyes will move with the head. This movement (unlike the vestibulo-ocular reflex) is immediate and does not need sustained observation, and therefore may be repeated a few times in each direction. It is advisable to disconnect the ventilator before doing this/or secure endotracheal tube with fingers as well because as sudden movements of the head while connected to the ventilator might result in tube displacement. (*See* Chapter 8 for demonstration **Figure 2**)

v. *Absence of gag reflex:* A firm swab (like an ear bud) can be used to firmly stimulate the posterior oropharynx on each side. This is done using tongue depressor and torch to look carefully for any arching movement of soft pallet when the swab is touched on each side of the posterior pharynx. The reflex should be absent. (*See* Chapter 8 for demonstration **Figure 6**)

vi. *Absence of cough reflex:* A suction catheter is passed through the endotracheal tube to gently stimulate the trachea. Usually, practice is to go up to a point where a resistance is felt and the gently suction out. Keep patients torso adequately exposed (covering the breast in female patients) to look for any cough reflex. The reflex should be absent.

3. *Documentation of apnea:* The aim of the test is to establish death of the respiratory center in the medulla. This is the last test done as the rise in carbon dioxide could damage the brain of a patient who is not brain dead and should therefore be done only after all other clinical tests for brain function are negative. The test itself involves disconnecting the ventilator for sufficient time for the partial pressure of carbon dioxide (pCO_2) in arterial blood to rise to 60 mm Hg. The test has to be aborted if the patient develops hypoxia, hypotension, or cardiac arrhythmia. There are several preventable errors that are made during the performance of this test. Some hints for performing the test successfully are:

- Reduce the respiratory rate by about 30% and increase the fraction of inspired oxygen (FiO_2) to 100% when beginning to do the clinical testing. The partial pressure of oxygen (pO_2) will be high and pCO_2 should be 40–45 mm Hg at the beginning of the apnea test.
- To prevent hypoxia during the period of apnea, a catheter is inserted into the endotracheal tube to provide oxygen. The length of insertion should be measured so that the catheter just protrudes beyond the endotracheal tube—too short and the oxygen will flow back, too long and it will be delivered into a bronchus. A flow >8–10 L/min might produce an element of ventilation and slow the rise of carbon dioxide. Hence, in most patients 4–5 L/min of oxygen flow is enough.
- The patient's volume status must be good. Carbon dioxide is a powerful systemic vasodilator, and as the pCO_2 builds during the apnea test, the patient will develop hypotension if the patient is not adequately hydrated. In patients with chronic airway disease or severe chest trauma, the apnea test may be difficult to perform.
- Every minute of apnea will rise the $PaCO_2$ by around 3 mm. Hence, an apnea of around 8–10 minutes may be needed to demonstrate a rise of CO_2 above 60 mm Hg from the baseline of 40–45 mm Hg (which is normal).
- In patients who are CO_2 retainers and a baseline $PaCO_2$ cannot be achieved, in them a rise of 20 mm above their baseline can be considered as a positive apnea test.
- There should be *no* breathing detected during the test.

Once all the above tests are done, then the patient is a potential brain dead patient. In Indian law, the same set of tests need to be done twice by two different doctors and only at the end of second clinical testing, the patient is declared dead by neurological criteria. Asking for an organ donation can be considered and should be made after declaring death.

It is important to decouple brain death from organ donation. Quite often brain death is only considered in context of organ donation. This prevents patients been declared dead and quite often continued on ventilator without any chance of recovery.

ORGAN DONOR MAINTENANCE

A brain dead donor is very labile as there is no neurological system intact to support hemodynamic as well as other systems such as urine output, temperature regulation, etc. Hence, it is imperative that as soon as a patient is identified as brain death or is potentially brain death, the donor maintenance as per the Indian Society of Critical Care Medicine (ISCCM) consensus statement protocol should be initiated. In clinical practice, it is important to start a potential donor maintenance program once a patient has a GCS of ≤5, absent eye signs or abnormal eye signs, and is ventilated with no clinical evidence of ventilator trigger from spontaneous respiration.

SUMMARY

The chapter covers in detail how to examine a patient who is potentially brain dead. In clinical situation, it is important to first rule our clinical mimics and confounders. It is important to ensure that the clinical diagnosis supports possible progression to brain death and is supported by clinical history and CT/MRI of brain. A thorough examination is necessary and should be diligently carried out. Remember to decouple brain death from organ donation.

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Chapter

38

How will I Approach/Examine a Patient with New-Onset Seizure and Status Epilepticus in the Intensive Care Unit?

Rohit Aravindakshan Kooloth, Nagarajan Ramakrishnan

ABSTRACT

Critically ill patients often present with seizures in the intensive care unit (ICU), which may pose a challenge to diagnose and treat. Early detection and treatment of seizures are of paramount importance to prevent any secondary poor outcomes and morbidity. Actively seizing patients who do not wake up appropriately despite termination of the tonic-clonic contractions should have an electroencephalogram (EEG) done early to titrate therapy. A rapid and focused examination of the patients with seizures is important to identify patients who have subtle seizures, causes for seizures, and complications. In the following chapter, we review the definitions, classification, etiopathogenesis, and evaluation of a patient presenting with seizures in the ICU. Furthermore, the readers will understand how to examine a patient with seizures.

INTRODUCTION

Seizures in the intensive care unit (ICU) have been reported in 3.3–34% in various studies. In the critical care setting, it may become difficult to recognize the seizures clinically, particularly in patients on mechanical ventilator support who are sedated and sometimes on paralytics. In patients whose sensorium is affected, history of prior seizures may not be available unless the family provides details of past medical history.

Status epilepticus may be the primary reason for admission to intensive care or may occur in any admitted ICU patient with other non-neurocritical illness for the first time. The importance of recognizing seizures in the ICU is due to high incidence of secondary brain injuries such as brain tissue hypoxia, tissue hypoperfusion, and excitotoxic damage due to recurrent seizures.

CASE SCENARIO

A 34-year-old male patient presented to the emergency department with a history of fever for 3 days and altered sensorium as described by the attender. On arrival, the patient started to have generalized tonic-clonic seizures. Vital signs recorded were heart rate of 134 beats/min, blood pressure (BP) 140/70 mm Hg, and saturation of 99% on room air. The temperature was 39.3°C for which paracetamol was administered. Patient was placed in the recovery position with supplemental oxygen and continued to have seizures despite the loading dose of benzodiazepine. The seizures continued to last >5 minutes despite loading with fosphenytoin, and eventually patient was intubated for airway protection and mechanically ventilated requiring propofol for sedation and rocuronium for paralysis. A computed tomography (CT) scan of the brain did not reveal any acute changes or evidence of cerebral edema.

The patient was shifted to the ICU and lumbar puncture was done, which showed increased protein and cell count (predominantly lymphocytes). Cerebrospinal fluid (CSF) blood sugars were within normal limits. Patient was started on empirical acyclovir. An EEG was done bedside to achieve burst suppression with propofol and fosphenytoin along with levetiracetam.

DEFINITION AND CLASSIFICATION

Definitions

A *seizure* is a sudden change in the behavior of an individual due to electrical hypersynchronization of neuronal connections in the cerebral cortex. It may be secondary to a brain insult or occur unprovoked.

Epilepsy can be described when any of the following exists:

- At least two unprovoked seizures occurring >24 hours apart

- One unprovoked seizure and the likelihood of further episodes similar to the general recurrence risk after two unprovoked seizures (e.g., >60%) occurring over the next 10 years (e.g., stroke)

Status epilepticus is an electroclinical condition characterized by generalized tonic-clonic convulsions (GTCS) lasting continuously for >5 minutes or >2 discrete seizures without a recovery of consciousness in between.

The International League Against Epilepsy (ILAE) have incorporated two time points, t1 and t2, in order to conceptually define status epilepticus as follows:

- The first (t1) is the time at which an ongoing seizure activity should be regarded as abnormally prolonged, unlikely to stop spontaneously, and when treatment for status epilepticus should be started.
- The second (t2) is the time after which the ongoing seizure activity poses a significant risk of long-term complications.

The ILAE definition is as follows: "Status epilepticus is a condition resulting from either the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms that lead to abnormally prolonged seizures (after time point t1). It is a condition that can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures."

Refractory status epilepticus is defined as status epilepticus that does not terminate despite maximal benzodiazepine dose and adequately dosed long-acting antiepileptic (e.g., fosphenytoin).

Super-refractory status epilepticus is defined as status epilepticus recurring or persisting after at least 24 hours of deep sedation and antiseizure drugs, usually midazolam, pentobarbital, or propofol.

Classification

Seizures and status epilepticus may be categorized based on whether the seizure is focal or generalized. Most of the generalized tonic-clonic seizures may begin as a focal seizure (secondary GTCS) and may be difficult to distinctly categorize.

Both focal and generalized seizures can be further divided if the clinical seizure activity is convulsive or nonconvulsive. The classification of the seizures is important to determine the morbidity and to determine the need for aggressive treatment.

ETIOLOGY

Most seizure or status epilepticus encountered in the ICU are generally secondary to intracranial pathology or a toxic or metabolic disturbance.

- *Acute structural brain injury:* Stroke, subarachnoid hemorrhage, traumatic brain injury, cerebral hypoxia, infection (encephalitis, meningitis, and abscess), or brain tumor
- Distant or long-standing structural brain injury (e.g., prior head injury or neurosurgery, perinatal cerebral ischemia, cortical malformations, arteriovenous malformations, and low-grade brain tumors)
- Nonadherence to antiepileptic medications in patients with prior epilepsy
- Withdrawal syndromes associated with the discontinuation of alcohol, barbiturates, or benzodiazepines
- *Metabolic abnormalities:* Hypoglycemia, hepatic encephalopathy, uremia, hyponatremia, hyperglycemia, hypocalcemia, hypomagnesemia, or sepsis
- Overdose or use of drugs that lower the seizure threshold, including theophylline, carbapenems (e.g., imipenem), high-dose penicillin G, cefepime, quinolone antibiotics, metronidazole, isoniazid, tricyclic antidepressants, bupropion, lithium, clozapine, flumazenil, bupivacaine, metrizamide, and dalfampridine.
- *Autoimmune causes:* More commonly recognized recently and generally present with status epilepticus. May sometimes also present with refractory status epilepticus and commonly associated with paraneoplastic syndromes [e.g., autoantibodies against neuronal proteins, such as the anti-leucine-rich glioma inactivated 1 (LGI1) protein, the N-methyl-D-aspartate (NMDA) receptor, the alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor, and the B1 subunit of the gamma-amino butyric acid B (GABA-B) receptor]; others include multiple sclerosis, adult-onset Rasmussen encephalitis, Hashimoto encephalitis, and lupus vasculitis.

PATHOPHYSIOLOGY

It has been generally accepted that inflammation is not only a consequence of seizure activity but also may contribute to epileptogenesis. There is a neurochemical imbalance between the excitatory and inhibitory neurotransmitters. Glutamate is the most common excitatory neurotransmitter associated with NMDA receptor activation and GABA is the most common inhibitory neurotransmitter. Sustained seizure activity seen in status epilepticus reduces GABA inhibition. This leads to unopposed glutamate accumulation with NMDA activation, increasing the intracellular calcium ions, leading to cell death.

Significant physiological changes happen during seizures. The initial phase is due to catecholamine surge leading to tachycardia, hypertension, hyperthermia, with mixed acidosis. The late phase may be associated with a drop in BP. The clinical implications of the changes in a critically ill patient are that the patient may be intubated and paralyzed or remain comatose and continue to have electrical seizures only detected by EEG monitoring, which may have deleterious neurological effects. Moreover, the longer the seizure lasts, there may be a more beneficial role to use NMDA receptor blockers such as ketamine in the termination of the seizure activity.

DIFFERENTIAL DIAGNOSIS

Recognition of seizures and status epilepticus may not always be easy in the ICU setting, especially nonconvulsive status epilepticus (NCSE).

- *Psychogenic nonepileptic seizure and psychogenic status epilepticus* may present with bilateral extremity motor movements with some preservation of responsiveness. It may also be associated with forward pelvic thrusting and geotropic eye movements. Video and EEG monitoring are the best way to establish a diagnosis and it is important to recognize this as a differential as the patient may be subjected to unnecessary medications, which may lead to further complications.
- *Encephalopathies* are extremely common in the ICU setting, which may present with irregular movements, myoclonus, and unresponsiveness. It may be possible to distinguish only with EEG recording from a true seizure or status epilepticus.
- *Movement disorders* such as tremors and dystonia may mimic status epilepticus. Clinically, these are rarely associated with diminished responsiveness and are differentiated with the use of EEG. Dystonic posturing may be a part of sympathetic overactivity in an unresponsive patient with severe traumatic brain injury and intracranial hemorrhages.

HISTORY AND SYMPTOMATOLOGY

When assessing individuals who are experiencing their first seizures, ask the patient or a trustworthy observer for a thorough medical history. Symptoms including postevent amnesia, déjà vu, mood swings, limb jerking, tongue biting, and head twisting should be looked out for. A comprehensive history may reveal previously unrecognized seizures in many patients who have experienced their "first" seizure. It is important to rule out different etiologies. To distinguish seizures from other episodes (such as syncope), witness accounts are crucial. Remember that patients with new-onset or breakthrough seizures require appropriate assessment and quick follow-up. Detailed information is provided in **Table 1**.

Clinical Manifestations

Seizure recognition in the ICU is generally difficult due to the occurrence of nonconvulsive seizures in the setting of depressed consciousness due to various factors. They may be masked by the use of sedatives and paralytics used for concurrent critical illnesses such as severe adult respiratory distress syndrome (ARDS) and refractory intracranial hypertension.

Patients with *generalized convulsive status epilepticus* have obvious bilateral tonic and/or clonic motor activity and loss of consciousness that may lead to potentially serious complications and morbidity if persistent.

Focal motor status epilepticus is associated with focal regional jerky movements with loss of consciousness. Epilepsia partialis continua is a refractory form with retained awareness commonly due to inflammatory and infectious etiologies and neoplasms.

Myoclonic status epilepticus is characterized by focal or generalized myoclonic jerks. Following anoxia, the prognosis is generally fatal for patients with this disorder but with metabolic encephalopathies, it is often reversible.

Nonconvulsive status epilepticus may be focal or generalized and can occur in about 8–37% of the general critically ill patient population. It is defined as a persistent change in the level of consciousness, behavior, autonomic function, and sensorium from the baseline associated with epileptiform activity recorded in the EEG but without major motor signs. Time limits for the definition have not been determined but any activity on EEG lasting for >10 minutes is considered NCSE. There should be a high index of suspicion in patients with altered sensorium, unexplained stupor, or confusion in the ICU setting with no other cause.

GENERAL PHYSICAL EXAMINATION

A quick general physical examination is mandatory in all patients presenting to the ICU with seizure disorder.

TABLE 1: History and symptomatology—new-onset seizures.

Domain	***Relevant history to note***	***Comments***
Previous history and medication history	*Convulsions:* • Previous history of convulsions • Past CNS disorders—stroke/tumor/infections *Medications:* • Noncompliance • Drug overdose/poisoning *Substance abuse and withdrawal effects* (alcohol/benzodiazepines/cocaine)	Noncompliance to treatment is a common cause of breakthrough seizures in a known patient of epilepsy. Drug overdose is often missed (e.g., eucalyptus oil sniff). Certain drugs can lower seizure thresholds and precipitate seizures [e.g., antidepressants (bupropion), antibiotics (fluoroquinolones, imipenem, metronidazole, and isoniazid), opioid analgesics (tramadol), and immunosuppressants (tacrolimus)]
Consciousness	*Sensorium:* • Confusion—it can be post ictal • Comatose—nonconvulsive status, CVA, and CVT *Aura before convulsions*	Continuous nonconvulsive seizures may result in an appearance of confusion or a comatose state
Temporal events	*Current/recent illness:* • ENT infections, viral prodrome, illness in family, travel history • Trauma (head injury) • Self-harm, tablet consumptions, animal or insect bites • Sepsis *Recent surgery or instrumentation:* • Major surgery or minimally invasive	• Many times, temporal events give clue on the possible etiologies, for example, ENT infections left untreated could cause meningitis/cerebral abscess, venous sinus thrombosis • Travel history may indicate possibility of tropical infections • Animal bites may predispose to tetanus • Insect bites may result in severe allergic reactions
Convulsion specific	• Onset and offset—pattern of spread, for example, focal to generalized • Perioral twitches • Automatism and bizarre behaviors • History of fever/headache/blurred vision/vomiting—CNS infection • Headache/vomiting—SAH/tumor • New-onset neurological deficits—focal deficits/double vision/tinnitus	• This will help to differentiate type of seizure and its cause. Often difficult to recognize absence seizure • Complex partial seizures confused with psychiatric diseases • Relevant history to look for organic causes such as meningoencephalitis, subarachnoid hemorrhage, or intracranial space-occupying lesions
Systemic illness	• History of diabetes/glucose control—use of insulin • Uncontrolled hypertension • Chronic kidney disease • Renal failure—uremia • Electrolyte imbalance—hyponatremia, hypercalcemia • CTDs (SLE), CNS vasculitis, paraneoplastic syndromes	• Hypoglycemia is a common cause of seizures • Information regarding systemic diseases will help us to do focused evaluation and targeted further investigation • Hypertensive emergencies can present with PRES
Menstrual history	Pregnancy-associated seizures	Menstrual history in women of child-bearing age. Urine pregnancy test should be done in case suspicion arises

(CNS: central nervous system; CTS: connective tissue disorders; CVA: cerebrovascular accident; CVT: cortical vein thrombosis; ENT: ear nose throat; PRES: posterior reversible encephalopathy syndrome; SAH: subarachnoid hemorrhage; SLE: systemic lupus erythematosus)

Patients with NCSE may have episodic staring, nystagmus, clonic activity of the face and hand, and automatisms. They may be episodically unresponsive or alternately confused. Autonomic dysfunction in the form of tachyarrhythmias and fluctuating BP readings are seen commonly in sedated or comatose ICU patients with NCSE. Tachyarrhythmias are also common in drug intoxication such as tricyclic antidepressant overdose.

A macular skin rash may be present, which may point toward the etiology of autoimmune diseases such as systemic lupus erythematosus, Sjögren syndrome, or sarcoidosis. A rash may also be present due to antiepileptic drugs such as lamotrigine.

Severe hypothermia (rectal temperature <32°C) may point toward drug or alcohol intoxication or metabolic causes. Meningeal signs (e.g., nuchal rigidity) may point toward infective meningitis. A detailed local examination of the ear is important to rule out a potential source (acute suppurative otitis media and acute mastoiditis) for possible bacterial meningitis. Detailed information is provided in **Table 2**.

Neurological Examination

The level of consciousness in the ICU determines the structure of the neurological examination.

For a conscious patient, the clinician examines the higher mental functions (language, attention, memory, and orientation), cranial nerves, motor and sensory function, reflexes, and coordination to look for focal deficits that may point to the cause of the seizure. Commonly patients

TABLE 2: Summary of examination of patients with new-onset seizures or status epilepticus.

Domain	***Eliciting of signs/symptoms***	***Comments***
Head, face, and neck	• Confused state (postictal) • Periorbital twitching (absence seizures) • Facial symmetry (ischemic stroke or bleed) • *Rash:* Malar rash, viral exanthems, petechial rash in meningococcemia • Bruises, abrasions—suggestive of recent trauma • Surgical scars—previous surgeries for tumor/trauma • *Mouth:* – Tongue bite – Bleeding gums (leukemia, TCP) – Oral ulcers (vasculitis, CTDs) • *Eye:* Eyeball position (gaze preference), nystagmus (subtle seizure), unequal pupils, KF rings • *Fundus:* Systemic infections, choroid tubercles in TB, retinal hemorrhage, retinal infarction • *Cervical lymphadenopathy:* TB • Smell—peculiar smell may help to identify alcohol or toxins (OP)	This gives a quick clue for the presence of subtle signs of convulsions and regarding the etiologies like systemic diseases. Each examination is important to get a clue regarding: • Evidence of convulsion • Subtle signs of convulsions and NCSE • Causes for convulsions
Extremities	• *Upper limb and lower limb:* – Ongoing convulsions – Hemiparesis, plantar reflex (postictal/brain lesion) – Injection marks – Bruises and abrasions—recent trauma • *Fingers and palms:* – *Nail and digits:* Splinter hemorrhages, digital infarcts—IE, vasculitis – Clubbing—CLD, hepatic encephalopathy	The general examination may throw light on the cause of status epilepticus. Peripheral signs of vasculitis and infective endocarditis are easy to pick up
Trunk, back, and perineum	• *Rash:* Rask on trunk—herpes (simplex, zoster) • Genital ulcers—systemic diseases, infection • Skin pigmentations/nodules—neurofibromatosis • Eschars—scrub typhus-related CNS vasculitis/encephalopathy	The perineal and back examination is often missed in ICU patients that may give clue of source of sepsis
Bedspace and documents	• *Bedside EEG:* Diagnose NCSE and guide therapy • Vasopressor use indicates severe systemic disease and sympathetic failure • *Propofol, midazolam infusions:* In refractory SE • Blood sugars—hypo/hyperglycemias • Urine bag—color, quantity, trend of urine per hour—dark urine turning color with sunlight (porphyria)	A careful bedspace examination is helpful in getting the trend and severity of the condition

Contd...

Contd...

Domain	***Eliciting of signs/symptoms***	***Comments***
Cardiovascular	• Tachy or bradyarrythmias, irregular HR (atrial fibrillation and embolic stroke) • Fluctuating HR, BP—autonomic dysfunction—sign of NCSE • *BP:* Hypertension (PRES) whereas shock—effect of CNS dysfunction • *ECHO:* Vegetations/clots—embolic showers	• Look for signs that give clues of cause of embolic stroke, signs of NCSE • A quick echocardiography is helpful—vegetations, clots
Central nervous system	• Altered sensorium • Neurological deficits—cranial nerve palsies—can be transient and postictal or clue for further evaluation • Signs of peripheral neuropathy—involuntary movements. Twitching/ fasciculations (autoimmune) and sensory symptoms	It is common to have altered sensorium in patients with seizures. It may be postictal, continuation of convulsions (SE or NCSE), or part of CNS pathology (encephalitis viral/ autoimmune, tumors)
Other systems	• Look for septic focus (lung and GI) • *Liver failure:* CLD and HE, Wilson • *Hematology:* Poisoning (hemolysis, DIC, and coagulopathy), cytopenias—CTDs, drugs, HUS–TTP	Source of sepsis, clue for systemic diseases

(BP: blood pressure; CLD: chronic liver disease; CNS: central nervous system; CTS: connective tissue disorders; DIC: disseminated intravascular coagulation; ECHO: echocardiography; EEG: electroencephalogram; GI: gastrointestinal tract; HR: heart rate; HUS: hemolytic uremic syndrome; IE: infective endocarditis; KF: Kayser Fleischer; NCSE: nonconvulsive status epilepticus; OP: organophosphorus; PRES: posterior reversible encephalopathy syndrome; SE: status epilepticus; SLE: systemic lupus erythematosus; TB: tuberculosis; TCP: thrombocytopenia; TTP: thrombotic thrombocytopenic purpura)

TABLE 3: Medical Research Council sum score.

Score for each movement:
0 = No visible contraction
1 = Visible muscle contraction, but no limb movement
2 = Active movement, but not against gravity
3 = Active movement against gravity
4 = Active against gravity and resistance
5 = Active movement against full resistance

Movement tested

Upper limbs	***Lower limbs***
Wrist flexion	Ankle dorsiflexion
Forearm flexion	Knee extension
Shoulder abduction	Hip flexion

0 to 36: Quadriplegia to severe quadriparesis
36 to 48: Mild quadriparesis
48 to 60: Normal strength

have an extensor plantar response in the postictal phase. Muscular strength is generally tested using the validated Medical Research Council (MRC) scale **(Table 3)**.

For a comatose patient, level of consciousness, brainstem function, motor responses, and respiratory pattern are examined. Numerical scales used for this purpose are the Glasgow Coma Scale (GCS) or the full outline of unresponsiveness (FOUR) scale **(Table 4)**.

Brainstem function comprises pupillary light reflex, corneal reflex, oculocephalic reflex, oculovestibular reflex, and cough and gag reflex. Pupillary signs such as dilatation may point toward acute drug intoxication with cocaine abuse and pinpoint pupils seen in acute barbiturate poisoning and tricyclic antidepressant overdose.

There may be distinctive respiratory patterns, which may point out to the level of the lesion that may be causing the seizure episode. Bilateral hemispheric and systemic disorders may lead to crescendo-decrescendo pattern with apnea (Cheyne–Stokes respiration). Lesions of the midbrain and pons may present as tachypnea and respiratory alkalosis. Lesions of the inferior pons may present as apneustic pattern (2 to 3 seconds pause with full inspiration). Intrinsic and compressive lesions of the medulla may cause chaotic breathing of varying depth and rate (Biot breathing).

DIAGNOSTIC APPROACH (FLOWCHART 1)

History: It is important to collect prior history to the seizure episode if not witnessed although this may not be possible in an ICU setting. Triggers may include strong emotions, fever, lack of sleep, pregnancy, and stress **(Table 1)**.

Drugs are common offenders of causing seizures, moreover in patients with hepatic and renal

TABLE 4: Glasgow Coma Scale and full outline of unresponsiveness (FOUR) score.

Parameters	***Glasgow Coma Scale***	***FOUR score***
Eye response	4 = Eyes open spontaneously 3 = Eyes opening to verbal command 2 = Eyes opening to pain 1 = No eyes opening	4 = Eyelids open or opened, tracking, or blinking to command 3 = Eyelids open but not tracking 2 = Eyelids closed but open to loud voice 1 = Eyelids closed but open to pain 0 = Eyelids remain closed with pain
Motor response	6 = Obeys commands 5 = Localizing pain 4 = Withdrawal from pain 3 = Flexion response to pain 2 = Extension response to pain 1 = No motor response	4 = Thumbs-up, fist, or peace sign 3 = Localizing to pain 2 = Flexion response to pain 1 = Extension response to pain 0 = No response to pain or generalized myoclonus status
Verbal response	5 = Oriented 4 = Confused 3 = Inappropriate words 2 = Incomprehensible sounds 1 = No verbal response	
Brainstem reflexes		4 = Pupil and corneal reflexes present 3 = One pupil wide and fixed 2 = Pupil or corneal reflexes absent 1 = Pupil and corneal reflexes absent 0 = Absent pupil, corneal and cough reflex
Respiration		4 = Not intubated, regular breathing pattern 3 = Not intubated, Cheyne–Stokes breathing pattern 2 = Not intubated, irregular breathing 1 = Breathes above ventilator rate 0 = Breathes at ventilator rate or apnea
Maximum–minimum	15–3	16–0

dysfunction. Carbapenems (imipenem) commonly and fluoroquinolones are known to lower the seizure threshold. Theophylline can precipitate seizures in higher concentrations. Alcohol withdrawal, acute cocaine, or methamphetamine intoxication can also cause seizures. If suspected a comprehensive blood toxicology panel should be ordered.

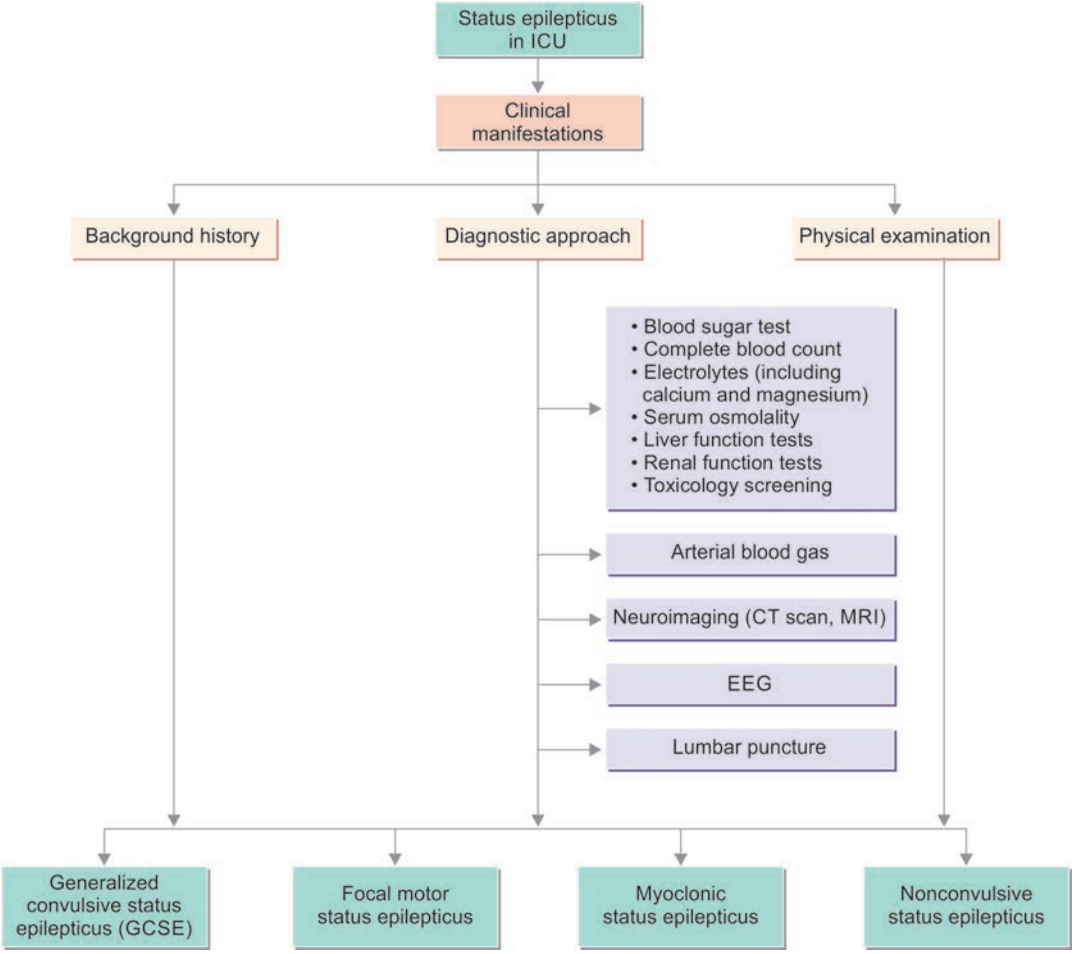
Metabolic screen: Serum glucose, osmolality, electrolyte levels (including ionized and total calcium with magnesium) along with liver and renal function tests should be routinely checked in all patients presenting with seizure. Arterial blood gas may be useful to check raised lactate levels and help in differentiating between syncope and psychogenic nonepileptic seizures. Routine complete blood count should be done to rule out infective etiologies.

Brain imaging: In critically ill patients with neurological pathology as primary or contributory cause for seizures, acute brain processes should be ruled out. CT scanning can be useful to detect acute blood, large tumors, or abscesses, edema, and large ischemic strokes. Magnetic resonance imaging (MRI) is helpful in detecting encephalitis, acute ischemic stroke, smaller tumors, and edema.

Electroencephalogram: Generally, in critically ill patients, there may be an urgent need to do an EEG to evaluate for subclinical or nonconvulsive seizure activity. Patients who develop refractory status epilepticus require continuous EEG monitoring. EEG may also be useful to differentiate between myoclonic status epilepticus **(Fig. 1)** from nonepileptic myoclonus.

The pattern of EEG findings such as burst suppression, post status epilepticus ictal discharges, and periodic lateralized epileptiform discharges during the initial 24 hours after control of status epilepticus were associated with poor outcome and raised mortality. Burst suppression secondary to pharmacological treatment for status epilepticus must be differentiated from burst suppression due to extensive cortical injury as both have different

Flowchart 1: Approach to seizures and status epilepticus in the ICU.


Status epilepticus in ICU
Clinical manifestations
Background history
Diagnostic approach
Physical examination
• Blood sugar test
• Complete blood count
• Electrolytes (including calcium and magnesium)
• Serum osmolality
• Liver function tests
• Renal function tests
• Toxicology screening
Arterial blood gas
Neuroimaging (CT scan, MRI)
EEG
Lumbar puncture
Generalized convulsive status epilepticus (GCSE)
Focal motor status epilepticus
Myoclonic status epilepticus
Nonconvulsive status epilepticus

(CT: computed tomography; EEG: electroencephalogram; ICU: intensive care unit; MRI: magnetic resonance imaging)

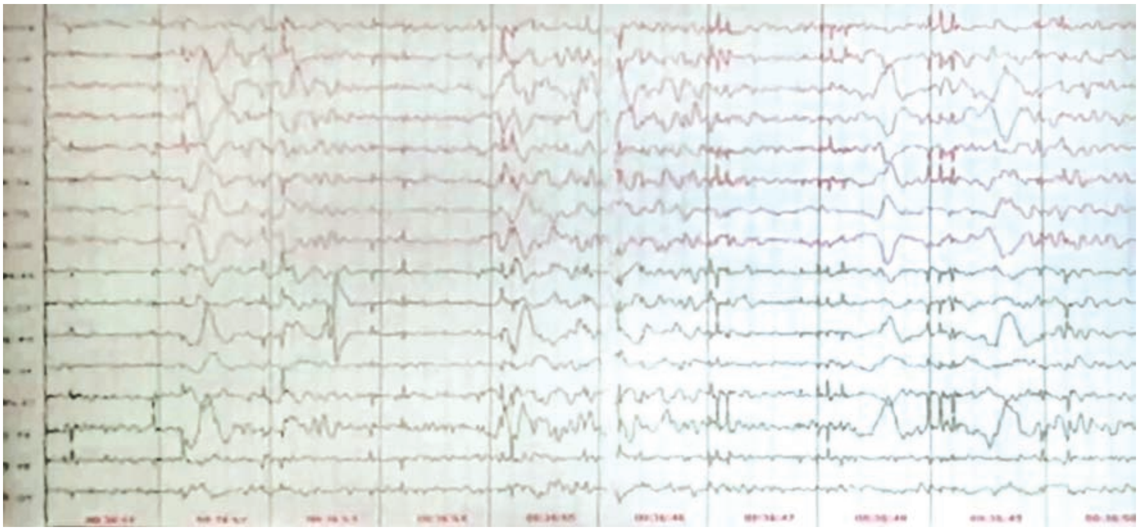


Fig. 1: Myoclonic status epilepticus on electroencephalogram (EEG) showing frequent bursts of rapid generalized polyspikes with a slow background in a patient with hypoxic ischemic encephalopathy in the intensive care unit (ICU).

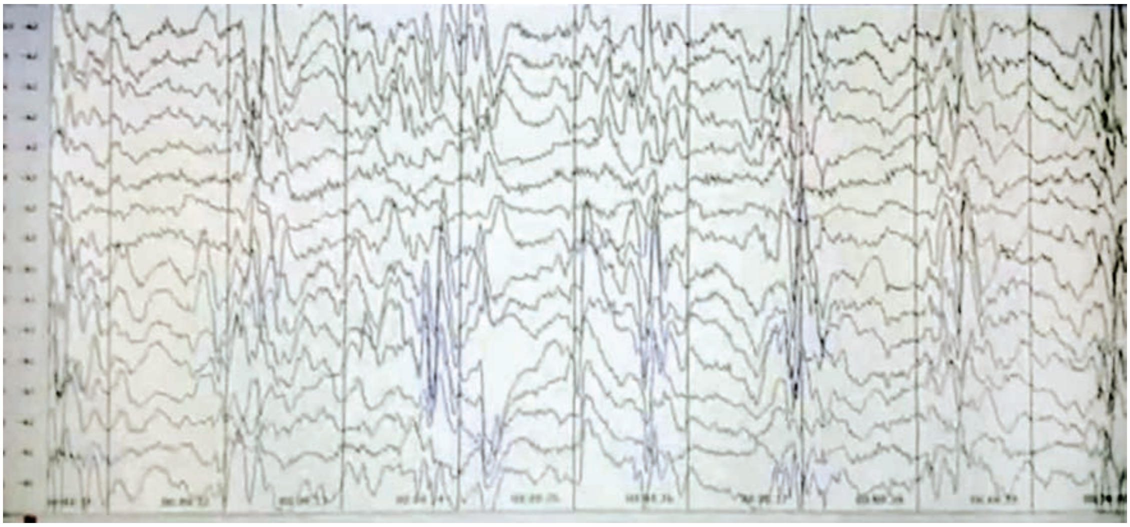


Fig. 2: An electroencephalogram (EEG) recording of nonconvulsive status epilepticus in a comatose intensive care unit (ICU) patient.

prognostic value. There may be a role in continuous EEG recording along with video monitoring in critically ill patients who remain comatose after clinical seizure, status epilepticus or in suspected nonconvulsive seizures **(Fig. 2)** with altered mental status. It may also be used to titrate intravenous medication to control seizures in refractory status epilepticus.

Lumbar puncture should be performed (after ruling out cerebral oedema) in patients in whom an infectious process involving the central nervous system is suspected. It may also be done in cases with leptomeningeal cancer and to detect autoimmune encephalitis.

SUMMARY

Treatment of seizures effectively in critically ill patients are relatively challenging due to complexities in drug dosing, need for EEG interpretation and expertise, and patient factors. Early recognition coupled with timely dosed antiepileptic medication remain the cornerstone to achieve optimal outcomes in the treatment of a critically ill patient presenting with acute onset seizures and status epilepticus.

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Chapter 39

Approach to Critically Ill Patients in Telemedicine

Rajesh Hiremath, Dileep Raman

ABSTRACT

Tele-intensive care unit (ICU) services represent the delivery of expertise to enhance critical care delivery, particularly in geographically isolated or resource-limited areas including large cities where intensivists may be distributed heterogeneously. By utilizing advanced telecommunication technologies, tele-ICUs enable continuous remote monitoring and expert consultation, thereby improving patient outcomes and optimizing ICU resource utilization. The use of tele-ICU services have shown to result in significant reductions in ICU mortality rates and length of stay, alongside increased adherence to best practice protocols. Key challenges include the adoption of technology in the form of electronic records, implementation, integration with existing software and data safety. The chapter discusses the step-by-step approaches with case examples towards setting up of tele-ICUs, protocols and workflows. There is emphasis on the critical role of tele-ICU services in enhancing patient care and the importance of continued innovation in critical care to solve for the triple challenge of balancing cost, quality and access.

INTRODUCTION

Telemedicine was started by US military and aerospace organizations around 1960 to 1970 to deliver medical services to remote areas. Telemedicine uses information technology tools to communicate, collect information, and deliver necessary care. Tele-intensive care unit (tele-ICU), in particular, is a subset of telemedicine that delivers the expertise of a specialist to remote areas, where the availability of specialists is constrained. Considerable experience now exists regarding the practice of telemedicine in high-acuity settings. Workflows and technologies have now become further refined to best suit different use cases and healthcare ecosystems. However, translation of traditional methods of care delivery to the technology-based models of care is often hard as many clinicians are only now getting accustomed to the tools at hand. Often, there is a perception that telemedicine is inherently inferior to standard care and that it cannot replace current practice. This view is rapidly changing as providers are beginning to realize that a hybrid approach is inevitable and potentially more patient safety and outcome centric. Telemedicine in acute care in certain circumstances may even be superior to routine care especially when related to speed of access and accountability. The bedside models and remote care constructs will likely complement each other strongly ensuring optimal care across geographies. In this chapter, we will discuss practical applications of remote care in this changing landscape and how it will likely to shape the future of critical care.

REMOTE MONITORING IN CRITICAL CARE

Remote monitoring of critical care patients augments the expertise of a bedside specialist. While this was historically the most critical function of remote care, namely, to allow access to a specialist during times of reduced staffing, today technology and workflow models allow much deeper integration of newer tools. Some of these models take aspects of remote monitoring and video streaming and combine them with software-based tools such as machine learning and assist in improved user experience to deliver a provider and patient experience that is seamless and efficient. There are two common models of care as follows:

1. 24×7 monitoring and management of patients by trained intensivists and critical care nurses. Such a

model uses an electronic medical record (EMR) for accessing and delivering information. This model also uses audio-visual data to visualize the patient and provide necessary inputs. The intensivist who is on call for the service is informed about the emergent problems and reactive or proactive services are provided. This model is round the clock and the providers may interact during the admission process and contribute to daily progress notes. They will also support during emergencies and family interactions. This is a "high touch" model and is also known as synchronous care.

2. In the second model, hospital-based telemonitoring systems may or may not use an EMR along with teleconferencing tools to provide services. Consultants are involved as and when required in a reactive framework. The workflow is generally used as a second opinion service or used when there is a need for an intensivist input. These are "light touch" models.

More pervasive ways of remote care augmented by technology are being operationalized in various health systems. They may function in a smart ICU paradigm that involves the integration of EMRs with intelligent decision support systems and patient monitoring devices. Such systems not only enhance remote monitoring but also strengthen traditional bedside teams. In these systems, the hospital has generally attained high levels of information systems integration that are certified by various bodies. Examples of this grading include the Healthcare Information and Management Systems Society (HIMSS) system (grades 1 to 7) and Electronic Medical Record Adoption Model (EMRAM) levels of EMR use. ICUs of 2030 and beyond will likely move in this direction where the entire infrastructure and workflows are just as amenable to bedside care as they are to remote care.

CHECKLIST FOR TELE-INTENSIVE CARE UNIT DEPLOYMENT

While the aspirational journey toward a smart ICU framework is ideal, resource-constrained settings may require a starting playbook to lower the barrier to receiving the care their patients need. To ensure a minimum level of operational success, a basic tele-ICU system must have certain prerequisites that have to be met by hospitals. They can broadly be divided into personnel, infrastructure, and processes requirements. A sample list that is representative is provided in **Boxes 1 to 3**.

BOX 1: Minimum personnel requirements at the bedside to deploy remote services in the hospital.

Personnel

- A trained person for intubation and to perform invasive procedures on call
- 1:4 nursing ratio for nonintubated cases per shifts
- 1:2 nursing ratio for noninvasive ventilation (NIV) or intubated cases per shift
- Two ward staff for every 10 beds per shift
- Information technology coordinator on call 24×7
- One senior nurse per shift must be the tele-ICU coordinator, an additional person to assist with data transfer may be needed on a case-by-case basis

BOX 2: Basic infrastructure required to start an intensive care unit (ICU) with tele-ICU support in the hospital.

Medical hardware and infrastructure

- Arterial blood gas (ABG) machine analyzer: 1 per ICU
- Portable X-ray machine: 1 per ICU
- Reliable oxygen supply
- 24×7 basic laboratory back-up
- Electrocardiogram (ECG) machine
- Infusion or syringe pumps
- Bedside monitors for each bed
- Broadband internet with a back-up (leased line if available is preferred)
- Reliable electrical power
- A laptop and handheld smartphones for nurses

Note: This is not an exhaustive list but a representative sample of essential hardware that is needed at the bedside.

BOX 3: Basic processes required to ensure transfer of data and workflow efficiency.

Process

- Laboratory reports available at optimal times
- Fixed time for MO/MD medical team to round with tele-ICU team
- Medication and input-output reconciliation twice a shift
- RMOs must coordinate with the tele-ICU team for any medical decisions. Coordinators may also be nonmedical personnel
- Instructions to the bedside nurse to follow our orders as we are an extension of the bedside team
- Access to radiology and labs through LIS or PACS

(ICU: intensive care unit; LIS: laboratory information systems; PACS: picture archiving and communication systems; MD: Doctor of Medicine; MO: Medical Officer; RMO: Registered Medical Officer)

TECHNOLOGIES FOR TELEMEDICINE

Technology is a critical foundational element of tele-ICUs services. Ideally in a tele-ICU system, EMRs, radiology image archiving and communication systems (PACS), integrated hemodynamic monitoring systems, and an audio-visual interface to communicate with the bedside team must all be on a single integrated platform. This is not the case today in most hospitals as the creation of the network was done piecemeal and not as a single deployment. There are also multiple peripheral hardware devices that are often not mandatory but assist in either patient examination or transfer of data.

TOOLS OF TELE-INTENSIVE CARE UNIT (TABLE 1)

Workflow

Tele-ICU workflow is considerably different from bedside workflow. However, the idea is not to replace or replicate bedside workflows but play to the strengths of the model, which is optimizing the reach of specialists. For this, the bedside team and the remote providers will need to see all bedside care data on the same platform that allows open access to medical records for the remote specialist and effective collaboration between providers. Key members of this team include the bedside nursing team, bedside registrar, primary consultant, teleregistered nurse (RN), telecritical care nurse (CCRN), nurse practitioner (NP) or physician extenders and remote consultants specialized in intensive care and even other specialties.

Tele-Intensive Care Unit Workflow

The remote providers must ideally be involved as soon as the patient enters the care area. For a critically ill patient, this may be the emergency department or the ICU. The bedside doctor examines the patients and gets a detailed summary while the bedside nursing team continues their routine plan of care. The key step is to now inform the remote nurse via an appropriate platform. The mode of communication may be electronic and even partially or completely automated. The automation will depend on the bedside team being on some form of the electronic record. If this does not exist, a circumstance common in resource-limited settings, the communication can be on the remote care platform. An electronic patient chart must be maintained on the platform and details such as vitals and documents (written notes, radiological reports, and laboratory reports) are uploaded on the platform. Bedside duty doctor may then call the tele-ICU consultant and give a summary about the patient. After a case discussion, along with visualization of the patient via PTZ (Pan Tilt Zoom) cameras, the teleconsultant documents a plan of action on the common platform. Teleconsultants may also conduct rounds with bedside duty doctors or primary consultants daily to assist with the plan of care.

TABLE 1: Common tools used in tele-intensive care units (ICUs).

Tools	*Usage*
Remote stethoscope	Cardiopulmonary sounds can be heard via a remote auscultation system connected to electronic stethoscopes. The utility in modern ICUs is limited due to the existence of better tools
Remote ultrasonography	Direct visualization of bedside ultrasounds by a remote team is possible allowing real-time assessment and decision-making
Pan-Tilt-Zoom (PTZ) camera, kiosk, tablet	Audiovisual experience allows detailed inspection of the patient. Electronic medical record (EMR) on tablet/phone, mobile kiosk allows easier bedside care and access to patient data
Remote patient monitoring (RPM)	Remote patient monitoring increases the effectiveness of the care delivered (3) by streaming physiological data to the remote provider

The platform must support various critical activities in the patient journey **(Fig. 1)**.

Vitals chart on EMR containing fields for key ICU data are shown in **Figure 2**. Admission notes on the EMR allow teams to follow a unified plan that is clear and auditable **(Fig. 3)**. Event notes on EMR supplement documentation for intermittent events that may not require a complete progress note **(Fig. 4)**. Medication administration records (MAR) maintain a log of events that happened after doctors' orders are placed. They denote "what actually happened" **(Fig. 5)**. The order section on EMR lists the doctor's orders. They denote "what must happen" **(Fig. 6)**. The laboratory section on EMR allows the providers to see laboratory data and other investigations **(Figs. 7 and 8)**.

Difference Between Remote Care Workflows and Conventional Workflows

Telephysicians act as an expert opinion or a second set of eyes in diverse settings. **Table 2** describes some common differences.

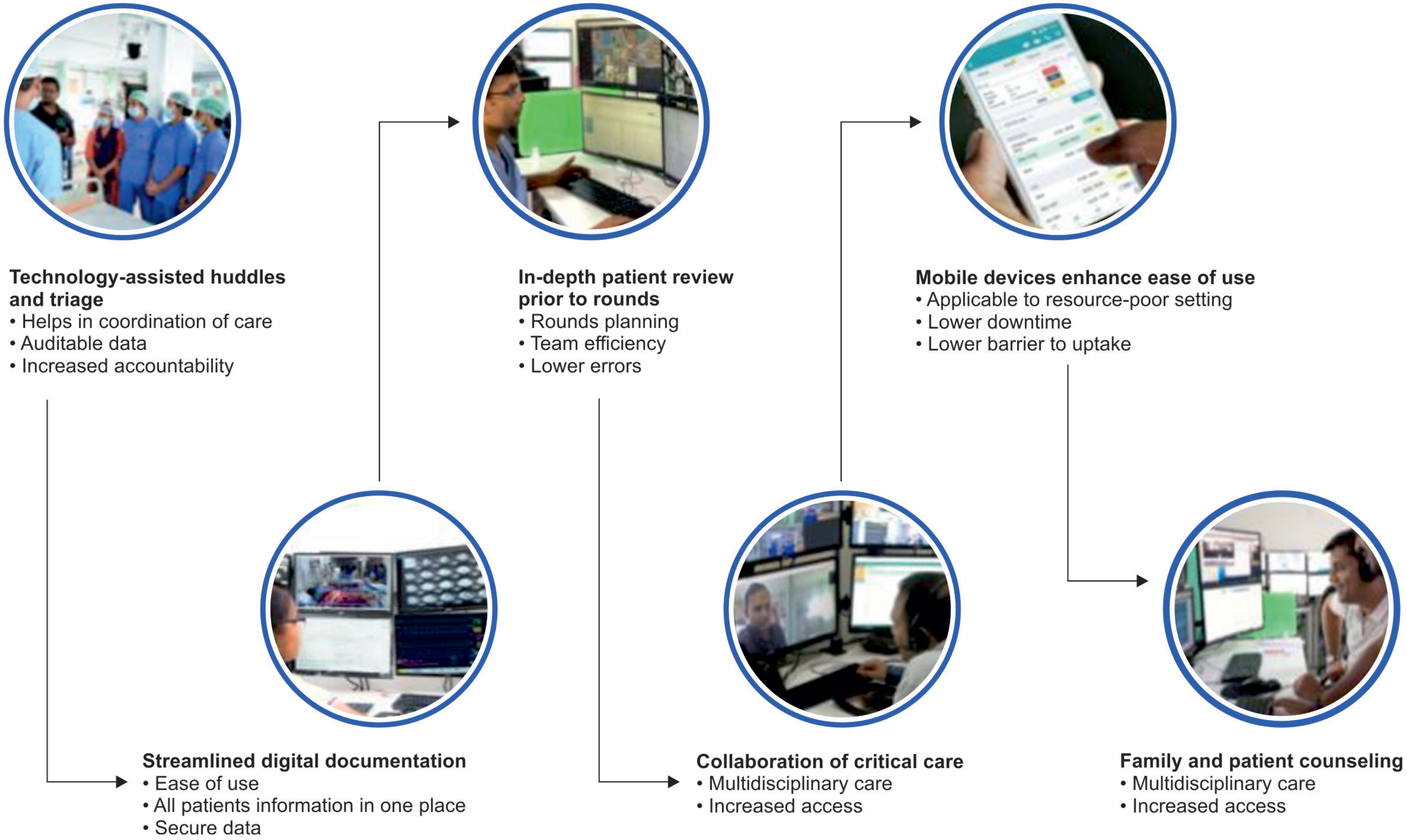
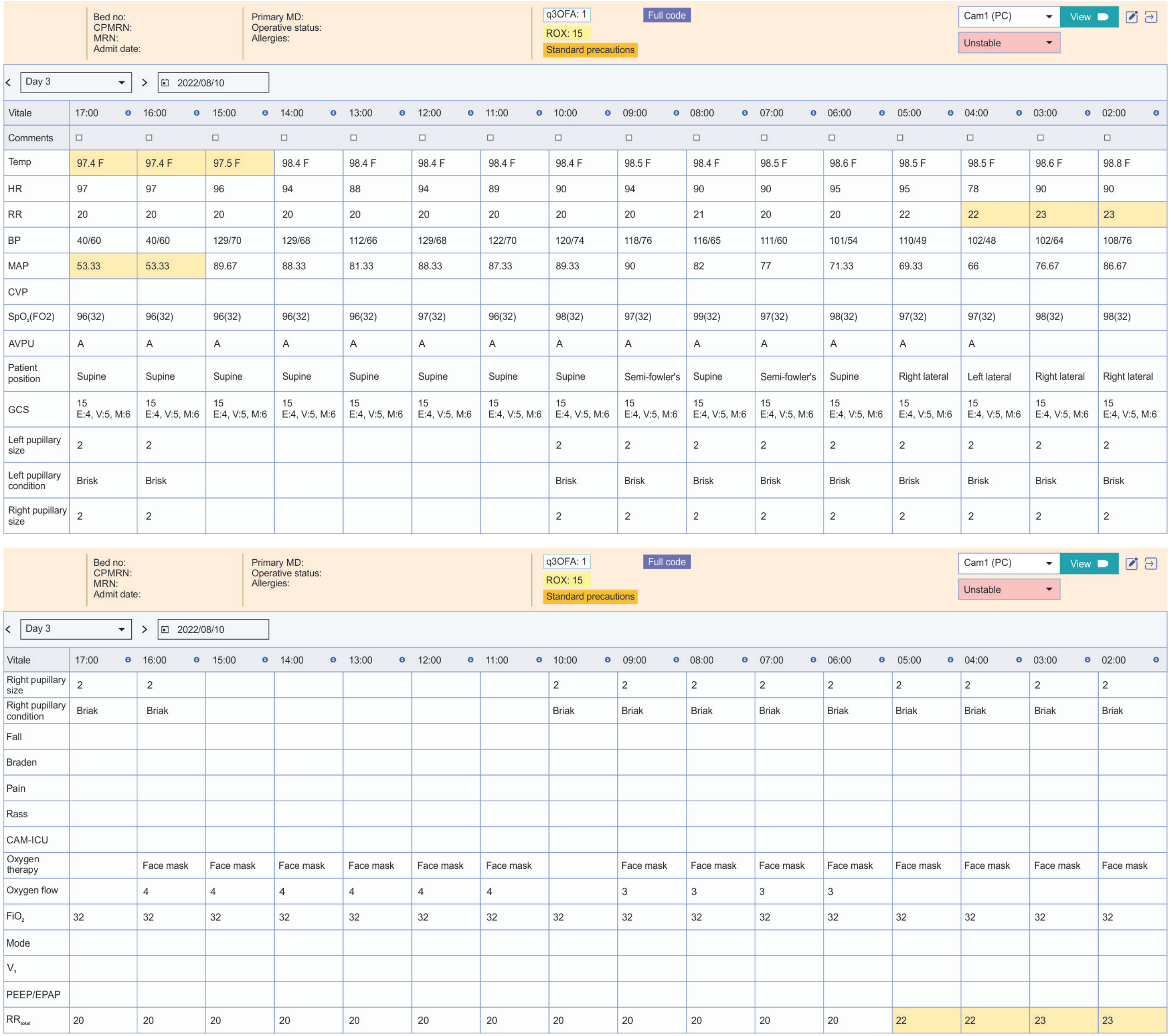

Technology-assisted huddles and triage
• Helps in coordination of care
• Auditable data
• Increased accountability
Streamlined digital documentation
• Ease of use
• All patients information in one place
• Secure data
In-depth patient review prior to rounds
• Rounds planning
• Team efficiency
• Lower errors
Collaboration of critical care
• Multidisciplinary care
• Increased access
Mobile devices enhance ease of use
• Applicable to resource-poor setting
• Lower downtime
• Lower barrier to uptake
Family and patient counseling
• Multidisciplinary care
• Increased access

Fig. 1: Schematic representation of a typical tele-intensive care unit (ICU) workflow.
Source: Tele-ICU workflow. Obtained from Cloudphysician Healthcare after permission.



Bed no:
CPMRN:
MRN:
Admit date:

Primary MD:
Operative status:
Allergies:

q3OFA: 1
ROX: 15
Standard precautions
Full code

Cam1 (PC) View
Unstable

< Day 3 > 2022/08/10

Vitale	17:00	16:00	15:00	14:00	13:00	12:00	11:00	10:00	09:00	08:00	07:00	06:00	05:00	04:00	03:00	02:00
Comments	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Temp	97.4 F	97.4 F	97.5 F	98.4 F	98.4 F	98.4 F	98.4 F	98.4 F	98.5 F	98.4 F	98.5 F	98.6 F	98.5 F	98.5 F	98.6 F	98.8 F
HR	97	97	96	94	88	94	89	90	94	90	90	95	95	78	90	90
RR	20	20	20	20	20	20	20	20	20	21	20	20	22	22	23	23
BP	40/60	40/60	129/70	129/68	112/66	129/68	122/70	120/74	118/76	116/65	111/60	101/54	110/49	102/48	102/64	108/76
MAP	53.33	53.33	89.67	88.33	81.33	88.33	87.33	89.33	90	82	77	71.33	69.33	66	76.67	86.67
CVP																
SpO_2(FO2)	96(32)	96(32)	96(32)	96(32)	96(32)	97(32)	96(32)	98(32)	97(32)	99(32)	97(32)	98(32)	97(32)	97(32)	98(32)	98(32)
AVPU	A	A	A	A	A	A	A	A	A	A	A	A	A	A		
Patient position	Supine	Supine	Supine	Supine	Supine	Supine	Supine	Supine	Semi-fowler's	Supine	Semi-fowler's	Supine	Right lateral	Left lateral	Right lateral	Right lateral
GCS	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6	15 E:4, V:5, M:6
Left pupillary size	2	2						2	2	2	2	2	2	2	2	2
Left pupillary condition	Brisk	Brisk						Brisk	Brisk	Brisk	Brisk	Brisk	Brisk	Brisk	Brisk	Brisk
Right pupillary size	2	2						2	2	2	2	2	2	2	2	2

Bed no:
CPMRN:
MRN:
Admit date:

Primary MD:
Operative status:
Allergies:

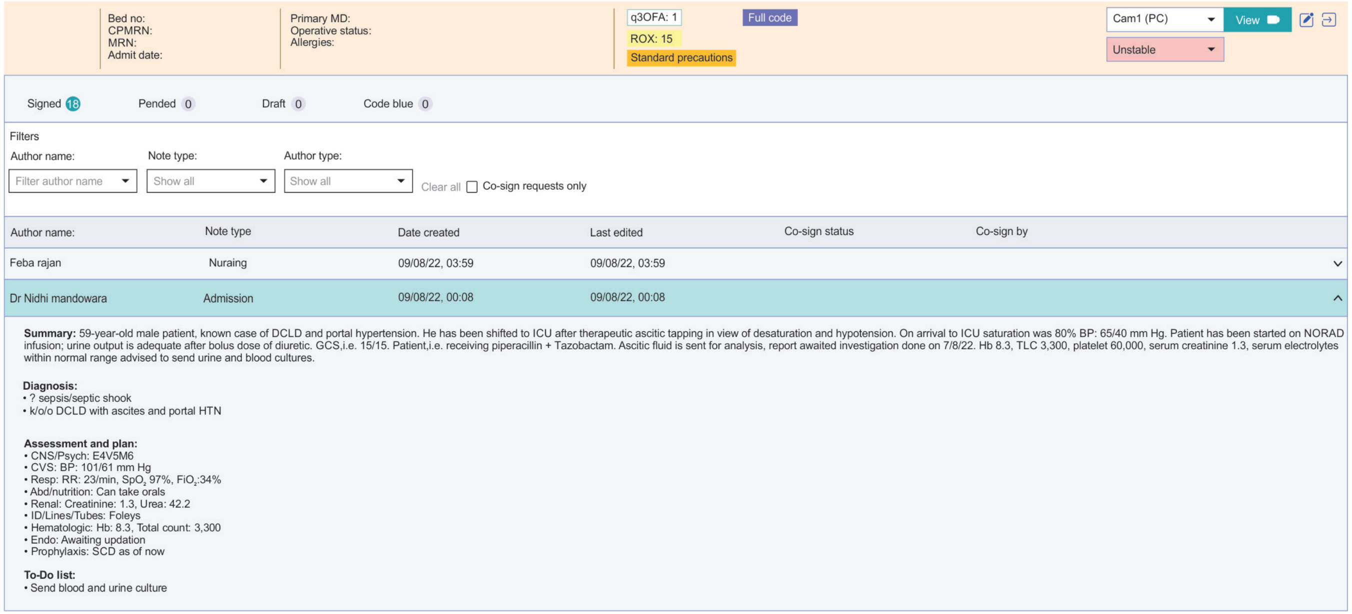
q3OFA: 1
ROX: 15
Standard precautions
Full code

Cam1 (PC) View
Unstable

< Day 3 > 2022/08/10

Vitale	17:00	16:00	15:00	14:00	13:00	12:00	11:00	10:00	09:00	08:00	07:00	06:00	05:00	04:00	03:00	02:00
Right pupillary size	2	2						2	2	2	2	2	2	2	2	2
Right pupillary condition	Briak	Briak						Briak	Briak	Briak	Briak	Briak	Briak	Briak	Briak	Briak
Fall																
Braden																
Pain																
Rass																
CAM-ICU																
Oxygen therapy		Face mask	Face mask	Face mask	Face mask	Face mask	Face mask		Face mask	Face mask	Face mask	Face mask	Face mask	Face mask	Face mask	Face mask
Oxygen flow		4	4	4	4	4	4		3	3	3	3				
FiO_2	32	32	32	32	32	32	32	32	32	32	32	32	32	32	32	32
Mode																
V_t																
PEEP/EPAP																
RR_{total}	20	20	20	20	20	20	20	20	20	20	20	20	22	22	23	23

Fig. 2: Vitals chart.
Source: Cloudphysician Healthcare; EMR–RADAR.


Bed no:
CPMRN:
MRN:
Admit date:
Primary MD:
Operative status:
Allergies:
q3OFA: 1
ROX: 15
Standard precautions
Full code
Cam1 (PC)
View
Unstable
Signed 18
Pended 0
Draft 0
Code blue 0
Filters
Author name:
Note type:
Author type:
Filter author name
Show all
Show all
Clear all
☐ Co-sign requests only

Author name:	Note type	Date created	Last edited	Co-sign status	Co-sign by
Feba rajan	Nuraing	09/08/22, 03:59	09/08/22, 03:59		
Dr Nidhi mandowara	Admission	09/08/22, 00:08	09/08/22, 00:08		

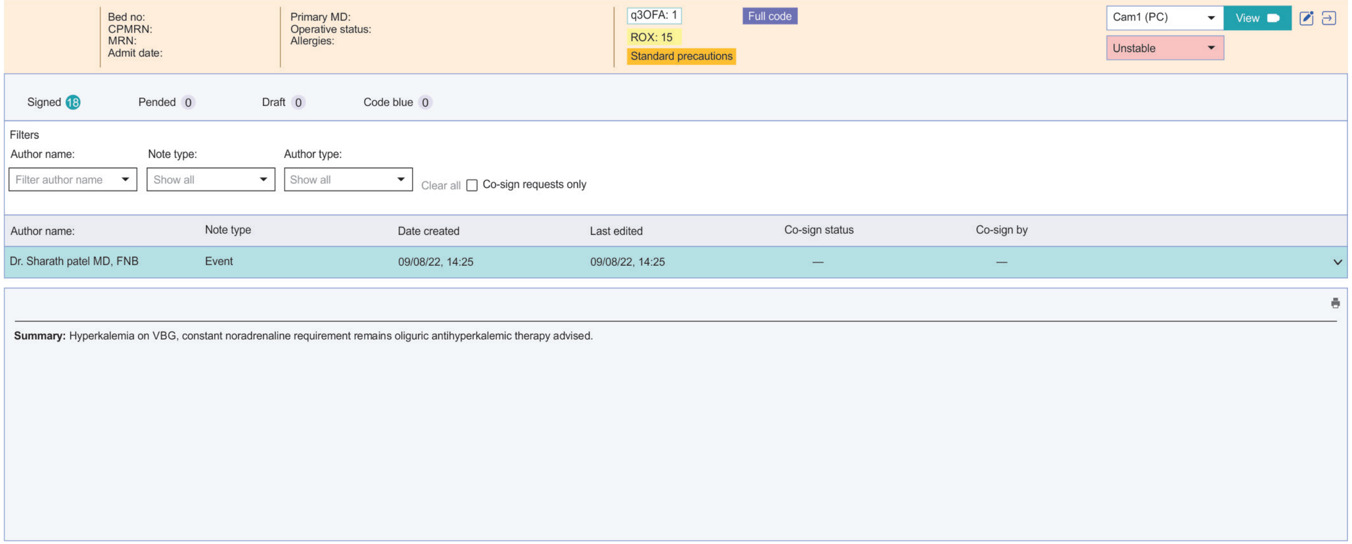
Summary: 59-year-old male patient, known case of DCLD and portal hypertension. He has been shifted to ICU after therapeutic ascitic tapping in view of desaturation and hypotension. On arrival to ICU saturation was 80% BP: 65/40 mm Hg. Patient has been started on NORAD infusion; urine output is adequate after bolus dose of diuretic. GCS,i.e. 15/15. Patient,i.e. receiving piperacillin + Tazobactam. Ascitic fluid is sent for analysis, report awaited investigation done on 7/8/22. Hb 8.3, TLC 3,300, platelet 60,000, serum creatinine 1.3, serum electrolytes within normal range advised to send urine and blood cultures.

Diagnosis:
- ? sepsis/septic shook
- k/o/o DCLD with ascites and portal HTN

Assessment and plan:
- CNS/Psych: E4V5M6
- CVS: BP: 101/61 mm Hg
- Resp: RR: 23/min, SpO_2 97%, FiO_2:34%
- Abd/nutrition: Can take orals
- Renal: Creatinine: 1.3, Urea: 42.2
- ID/Lines/Tubes: Foleys
- Hematologic: Hb: 8.3, Total count: 3,300
- Endo: Awaiting updation
- Prophylaxis: SCD as of now

To-Do list:
- Send blood and urine culture

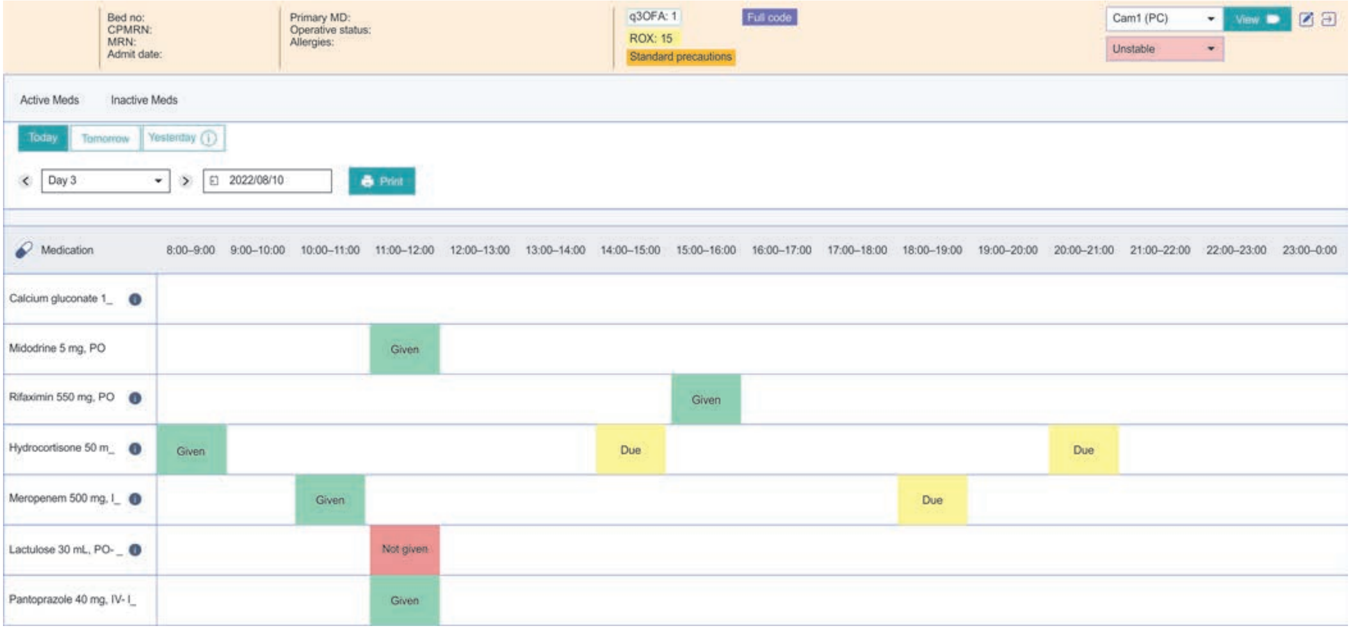
Fig. 3: Admission/progress notes.
Source: Cloudphysician Healthcare; EMR–RADAR.


Bed no:
CPMRN:
MRN:
Admit date:
Primary MD:
Operative status:
Allergies:
q3OFA: 1
ROX: 15
Standard precautions
Full code
Cam1 (PC)
View
Unstable
Signed 18
Pended 0
Draft 0
Code blue 0
Filters
Author name:
Note type:
Author type:
Filter author name
Show all
Show all
Clear all
☐ Co-sign requests only

Author name:	Note type	Date created	Last edited	Co-sign status	Co-sign by
Dr. Sharath patel MD, FNB	Event	09/08/22, 14:25	09/08/22, 14:25	—	—

Summary: Hyperkalemia on VBG, constant noradrenaline requirement remains oliguric antihyperkalemic therapy advised.

Fig. 4: Event notes.
Source: Cloudphysician Healthcare; EMR–RADAR.



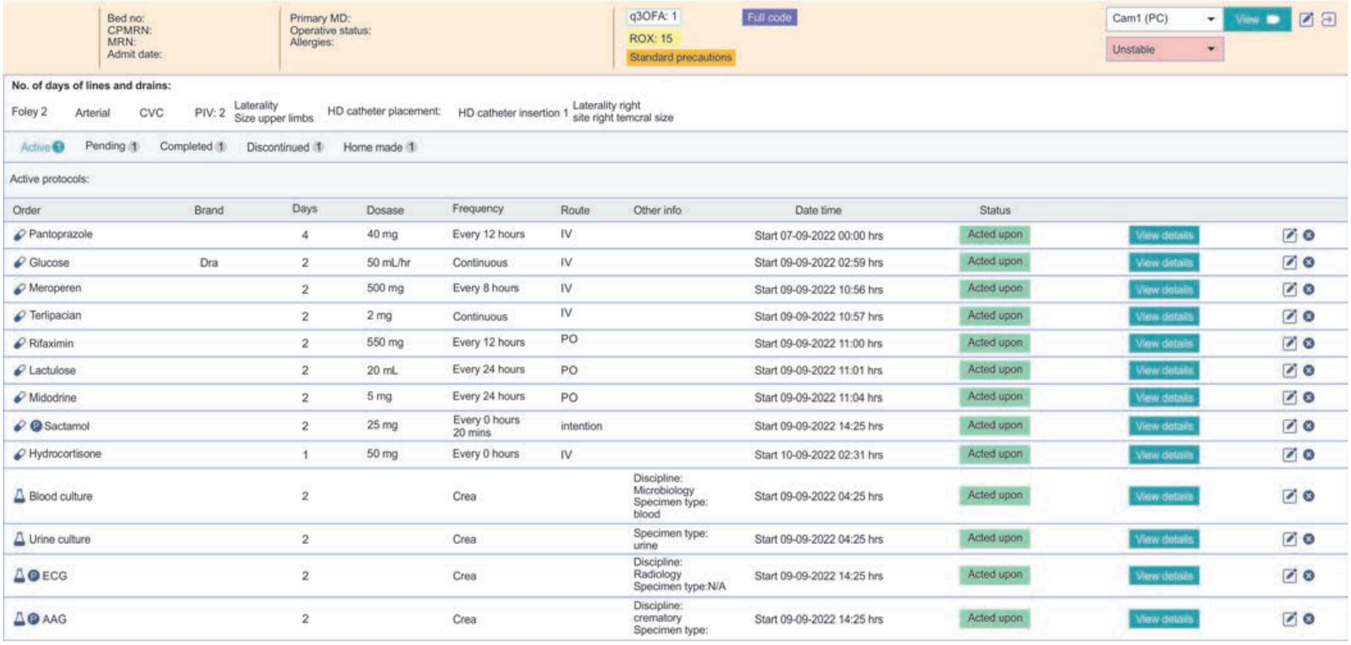
Bed no:
CPMRN:
MRN:
Admit date:

Primary MD:
Operative status:
Allergies:

q3OFA: 1
ROX: 15
Standard precautions

Medication	8:00–9:00	9:00–10:00	10:00–11:00	11:00–12:00	12:00–13:00	13:00–14:00	14:00–15:00	15:00–16:00	16:00–17:00	17:00–18:00	18:00–19:00	19:00–20:00	20:00–21:00	21:00–22:00	22:00–23:00	23:00–0:00
Calcium gluconate 1_																
Midodrine 5 mg, PO				Given												
Rifaximin 550 mg, PO								Given								
Hydrocortisone 50 m_	Given						Due						Due			
Meropenem 500 mg, I_			Given								Due					
Lactulose 30 mL, PO- _				Not given												
Pantoprazole 40 mg, IV- I_				Given												

Fig. 5: Medication administration record (MAR).
Source: Cloudphysician Healthcare; EMR–RADAR.



Bed no:
CPMRN:
MRN:
Admit date:

Primary MD:
Operative status:
Allergies:

q3OFA: 1
ROX: 15
Standard precautions

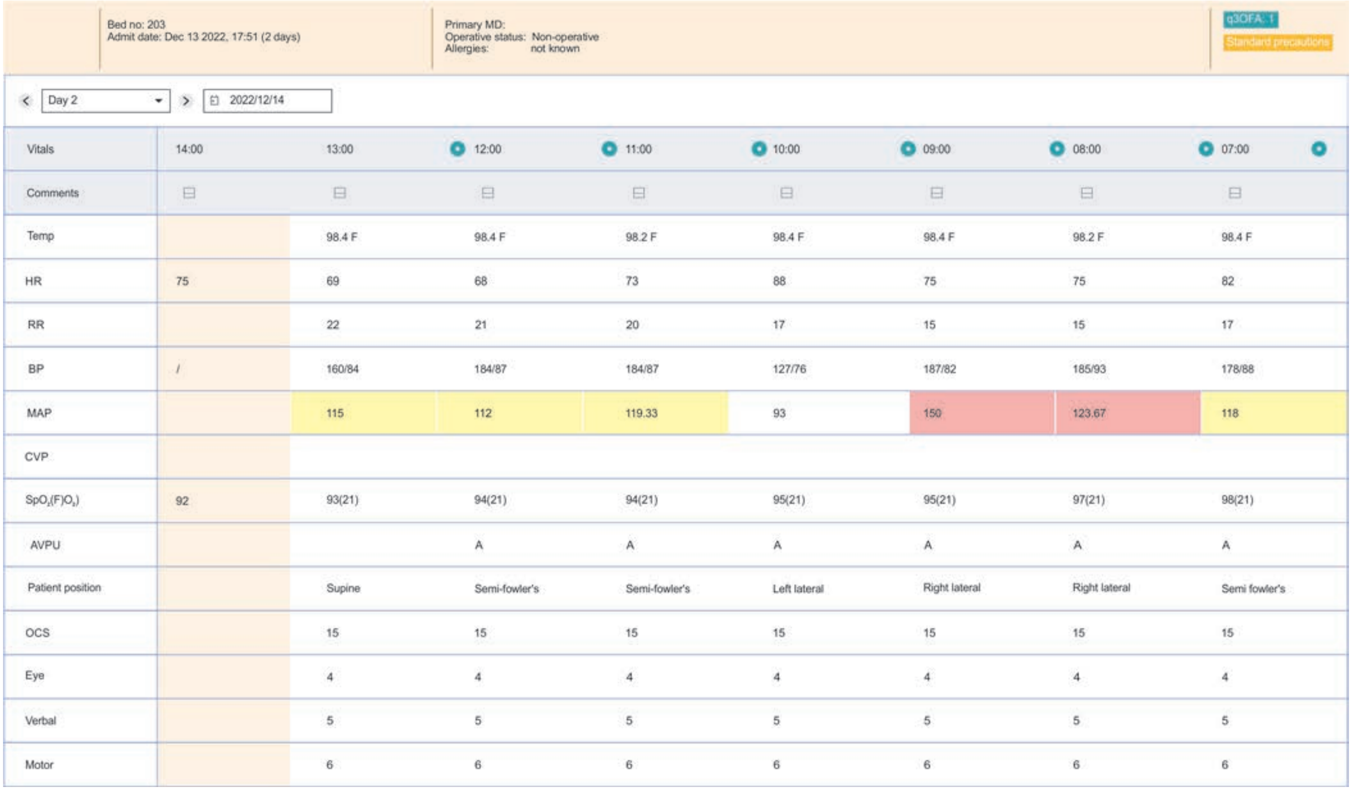
No. of days of lines and drains:

Foley 2 Arterial CVC PIV: 2 Laterality Size upper limbs HD catheter placement: HD catheter insertion 1 Laterality right site right temcral size

Active protocols:

Order	Brand	Days	Dosase	Frequency	Route	Other info	Date time	Status		
Pantoprazole		4	40 mg	Every 12 hours	IV		Start 07-09-2022 00:00 hrs	Acted upon	View details	
Glucose	Dra	2	50 mL/hr	Continuous	IV		Start 09-09-2022 02:59 hrs	Acted upon	View details	
Meroperen		2	500 mg	Every 8 hours	IV		Start 09-09-2022 10:56 hrs	Acted upon	View details	
Terlipacian		2	2 mg	Continuous	IV		Start 09-09-2022 10:57 hrs	Acted upon	View details	
Rifaximin		2	550 mg	Every 12 hours	PO		Start 09-09-2022 11:00 hrs	Acted upon	View details	
Lactulose		2	20 mL	Every 24 hours	PO		Start 09-09-2022 11:01 hrs	Acted upon	View details	
Midodrine		2	5 mg	Every 24 hours	PO		Start 09-09-2022 11:04 hrs	Acted upon	View details	
P Sactamol		2	25 mg	Every 0 hours 20 mins	intention		Start 09-09-2022 14:25 hrs	Acted upon	View details	
Hydrocortisone		1	50 mg	Every 0 hours	IV		Start 10-09-2022 02:31 hrs	Acted upon	View details	
Blood culture		2		Crea		Discipline: Microbiology Specimen type: blood	Start 09-09-2022 04:25 hrs	Acted upon	View details	
Urine culture		2		Crea		Specimen type: urine	Start 09-09-2022 04:25 hrs	Acted upon	View details	
P ECG		2		Crea		Discipline: Radiology Specimen type:N/A	Start 09-09-2022 14:25 hrs	Acted upon	View details	
P AAG		2		Crea		Discipline: crematory Specimen type:	Start 09-09-2022 14:25 hrs	Acted upon	View details	

Fig. 6: Medication chart.
Source: Cloudphysician Healthcare; EMR–RADAR.



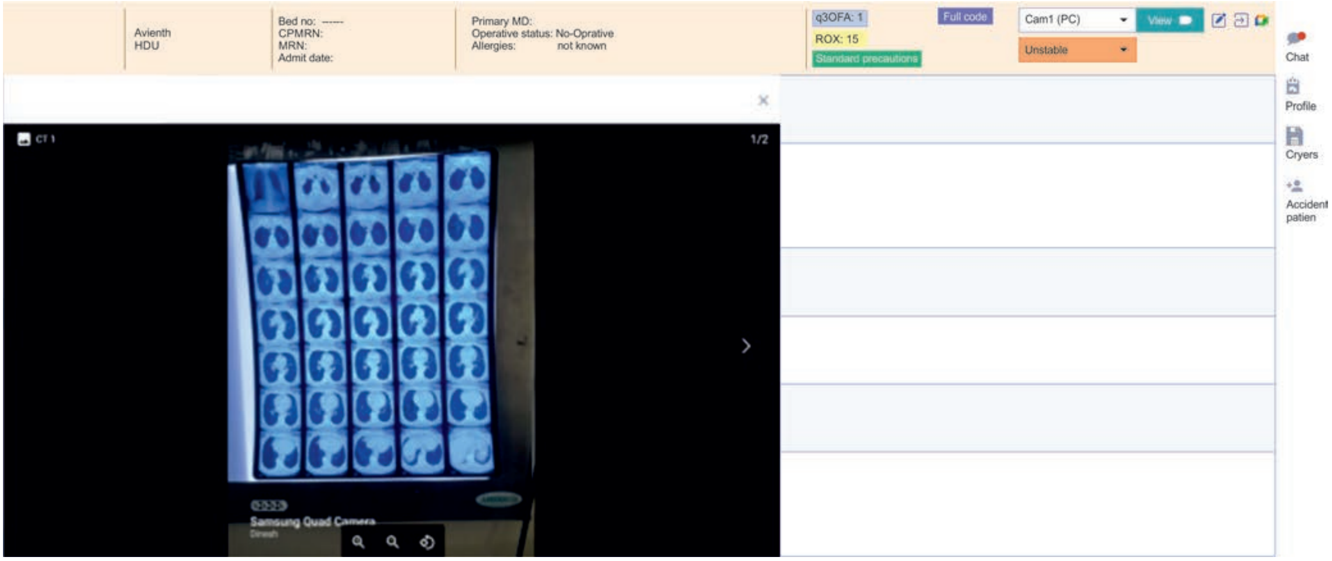
Bed no: 203
Admit date: Dec 13 2022, 17:51 (2 days)

Primary MD:
Operative status: Non-operative
Allergies: not known

q3OFA: 1
Standard precautions
Day 2
2022/12/14

Vitals	14:00	13:00	12:00	11:00	10:00	09:00	08:00	07:00
Comments	⊟	⊟	⊟	⊟	⊟	⊟	⊟	⊟
Temp		98.4 F	98.4 F	98.2 F	98.4 F	98.4 F	98.2 F	98.4 F
HR	75	69	68	73	88	75	75	82
RR		22	21	20	17	15	15	17
BP	/	160/84	184/87	184/87	127/76	187/82	185/93	178/88
MAP		115	112	119.33	93	150	123.67	118
CVP								
SpO$_2$(F)O$_2$)	92	93(21)	94(21)	94(21)	95(21)	95(21)	97(21)	98(21)
AVPU			A	A	A	A	A	A
Patient position		Supine	Semi-fowler's	Semi-fowler's	Left lateral	Right lateral	Right lateral	Semi fowler's
OCS		15	15	15	15	15	15	15
Eye		4	4	4	4	4	4	4
Verbal		5	5	5	5	5	5	5
Motor		6	6	6	6	6	6	6

Fig. 7: Vitals chart.
Source: Cloudphysician Healthcare; EMR–RADAR.



Avienth
HDU

Bed no: -----
CPMRN:
MRN:
Admit date:

Primary MD:
Operative status: No-Oprative
Allergies: not known

q3OFA: 1
ROX: 15

Standard precautions
Full code
Cam1 (PC)
View
Unstable
Chat
Profile
Cryers
Accident
patien
CT 1
1/2
Samsung Quad Camera

Fig. 8: Computed tomography (CT) films viewed on the electronic medical record (EMR) can assist with plan of care.
Source: Cloudphysician Healthcare; EMR–RADAR.

TABLE 2: Differences between tele-intensive care unit (ICU) augmented workflows and conventional workflows.

Tele-ICUs	***Conventional ICUs***
The first respondent is the duty doctor or the admitting consultant. Second review is by the remote consultant	The first response is either a duty doctor or primary consultant
The case is discussed with telephysicians ensuring specialist access	Case is discussed with primary consultants. If unavailable, care gaps are likely
Telephysicians give the management plan after discussing with the bedside duty doctor and camera visualization	The primary consultant examines the patient and makes a plan of management
Emergency situations can be addressed earlier and faster	Delays in managing the case as the primary consultant has to often examine the patient or may have incomplete data
It augments the ICU care at bedside	Specialist care is often compromised in resource-constrained settings
Communication pathway happens via duty doctor to the telephysician and then to the primary consultant. The platform ensures multiprovider collaboration	Communication pathway happens via duty doctor to the primary consultant

ASSESSING THE PATIENT

After the remote team is alerted to the arrival of a patient, assessment can begin. The presenting complaints might vary depending upon the situations. Patients can be classified into the following two categories:

1. Nonemergency
2. Emergency

Nonemergency

As the patient arrives in the emergency room, a duty doctor takes a brief history and does an examination. After going through the history and presenting complaints, the primary target organ system and initial differential diagnosis are identified. The duty doctor then discusses the case with the teleconsultant for a deeper review of possible diagnosis and management plans. The teleconsultant suggests the required investigations and plan of action, after going through the details of the findings made by the duty doctor. Please see case scenarios 1 and 2 for a more detailed discussion with examples.

Emergency

During emergencies, an immediate symptomatic approach is preferred. While an exhaustive analysis of various cases is not possible, some key organ system based examples are described below.

Central Nervous System

- *Abnormal sensorium:* Altered behavior is easily observed via the PTZ cameras. Remote patient physiological monitors may assist in suggesting possible therapies. Initial laboratory testing is also straightforward as urgent arterial blood gases (ABGs) and electrolytes can be ordered and reviewed enhancing the pace of care.
- *Hemiplegia/hemiparesis:* A brief history is often available initially, and the examination of the patient can be confirmed visually via a PTZ camera. It is possible to see eye movements, limb movement asymmetry, seizures, and abnormal movements. Certain cranial nerves can also be assessed remotely. Relevant investigations that are requested by telephysicians such as CT brain, magnetic resonance imaging (MRI) brain, for example, can be rapidly reviewed via PACS. For instance, if the CT images show a bleed, emergency neurosurgical intervention may be arranged without delay.
- *Seizures:* Seizures are often readily seen on PTZ cameras resulting in timely emergency medication administration. Relevant investigations can be expedited.

Cardiovascular System

- *Tachycardia/bradycardia:* Bedside monitor data is easier to review across a network with a connected care ecosystem resulting in common cardiovascular events being addressed with minimal delays. Telephysicians may then order a 12-lead electrocardiogram (ECG) and formulate a plan of action. Telephysicians also discuss the case with the primary physician and if required counsel the attenders at that point of time.

A monitor screen grab in the example **(Fig. 9)** shows a tachyarrhythmia. This was followed by an online discussion **(Fig. 10)** with the bedside team to quickly address the abnormality followed by the institution of appropriate therapy.

- *Hypotension/hypertension:* Similarly, events such as hypotension and hypertension **(Fig. 11)** are often picked up via PTZ camera by the remote team, which is then relayed to the bedside. Tele-ICU physicians may then assess the patient with the help of bedside nursing staff and will direct them regarding management of acute events and the orders are followed resulting in favorable outcomes.

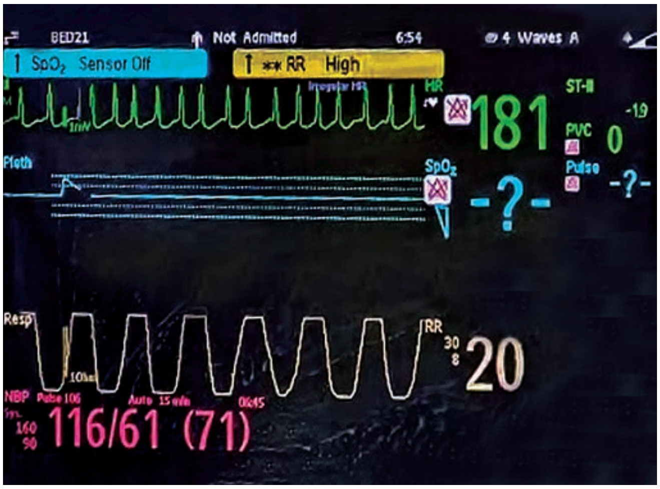


Fig. 9: Live view of the bedside monitor using HD, pan-tilt-zoom cameras.
Source: Care Centre, Cloudphysician Healthcare with permission.

- *ECG changes on monitor:* Critical ECG changes identified by remote services are informed to bedside nursing staff/registered medical practitioner (RMP). In **Figure 12**, the ST segment abnormalities resulted in the telephysician triggering a cardiac consultation and administration of antiplatelets without delay.
- *Cardiac arrest:* Patients who have an intrahospital cardiac arrest need cardiopulmonary resuscitation (CPR) per the Advanced Cardiovascular Life Support (ACLS) guidelines. Remote providers are ideally suited to act as timekeepers and team leads running the CODE BLUE. They can advise on drug administration and can check the quality of CPR **(Fig. 13)**. They can assist with the documentation of the event. This is especially important as the bedside team is often understaffed or busy.

Echocardiogram: Echocardiography is an ultrasound image that helps in assessing the function of the heart. A transthoracic echocardiogram is the preferred modality used to assess cardiac function in the ICU setting. Common echocardiographic views seen on transcardiac echocardiography are as follows:

- *Apical four-chamber view:* The patient is placed in a left lateral position. The transducer probe is placed

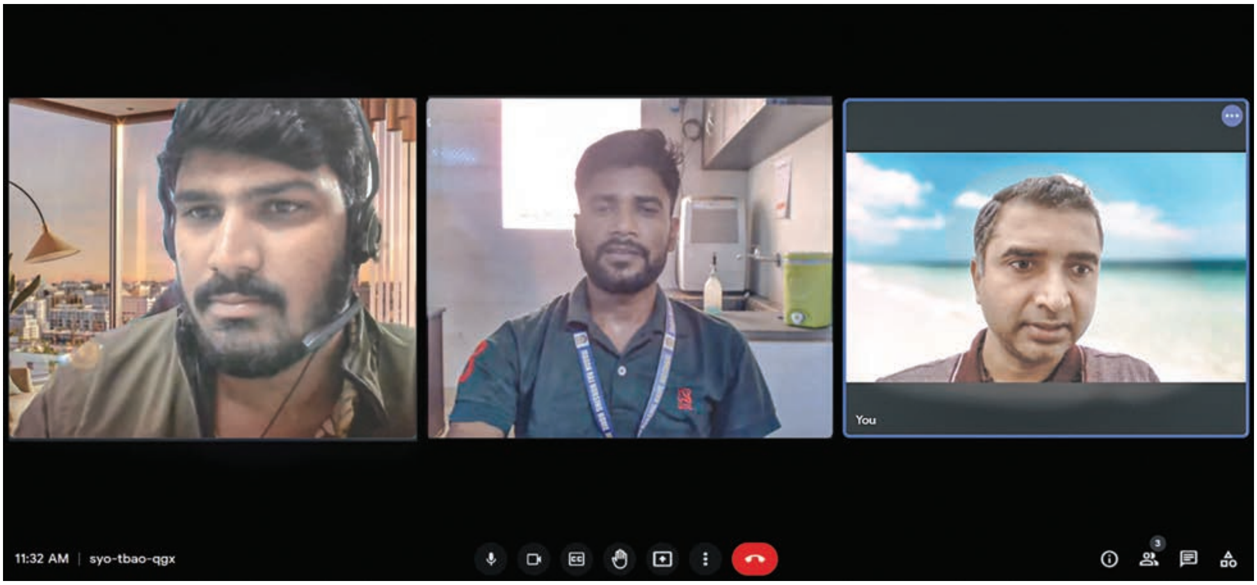


Fig. 10: Real-time telecommunication with the bedside.
Source: Care Centre, Cloudphysician Healthcare; Captured a real-time teleconversation with permission.

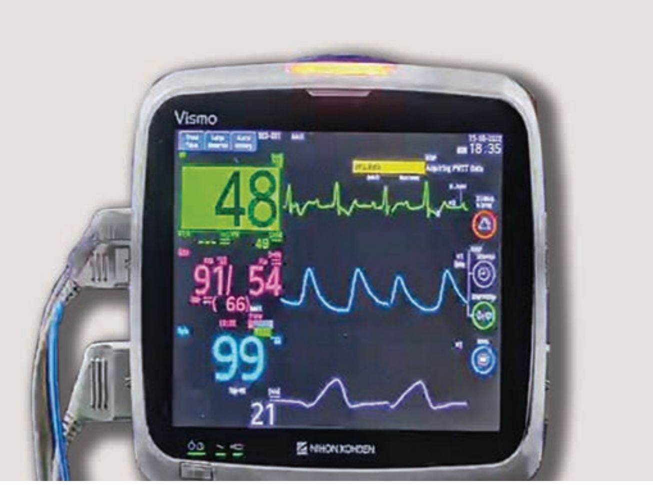


Fig. 11: Monitor with hypotension and bradycardia.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

Fig. 13: Supervised cardiopulmonary resuscitation (CPR) with remote physician as the team leader.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

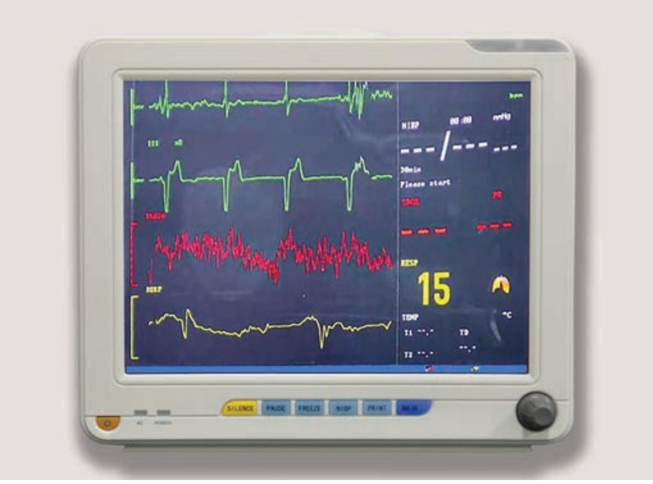


Fig. 12: Monitor showing ST changes.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

at the 4th intercostal space below the nipple and the probe pointer is angled toward the left side, slightly tilting the tail of the probe toward the left foot (horizontally). This position gives us a four-chamber view of the heart. The apical four-chamber view is a preferred view for hemodynamic assessment. This helps in assessing the dynamic functionality of cardiac outputs, valve regurgitations, and diastolic functions of cardiac chambers. This view also empowers us to view all chambers of the heart along with mitral and tricuspid valves. This view is most commonly used for hemodynamic assessment of the patient, which includes assessing diastolic dysfunction of the heart, valve regurgitations, and cardiac output. Structures assessed in this view include right ventricle, left ventricle, left atrium, right atrium, tricuspid valve, and mitral valve.

- *Parasternal long-axis view:* The transducer probe is placed at the 3rd or 4th left intercostal space adjacent to the sternum and with the probe pointing toward the right shoulder. Aortic and mitral valves and right and left chambers of the heart are best seen in this view.
- *Parasternal short-axis view:* From the parasternal long-axis view, rotate the transducer to 70–110° toward the left shoulder to get a better view of the aortic valve, mitral valve, right ventricle, left ventricle, and papillary muscles.
- *Subcostal view:* Transducer probe is placed immediately below or to the right side of the xiphoid process. The patient's abdomen has to be relaxed by flexing the knee and the probe pointer pointing toward the left elbow. Now tilt the tail of the probe toward the right foot (horizontally). It helps visualize the four chambers of the heart much better. It can be used as an alternative for apical view in cases of trauma and chronic obstructive pulmonary disease (COPD).

Figure 14 shows a bedside doctor performing an echocardiogram (apical view) after discussing with a teleconsultant.

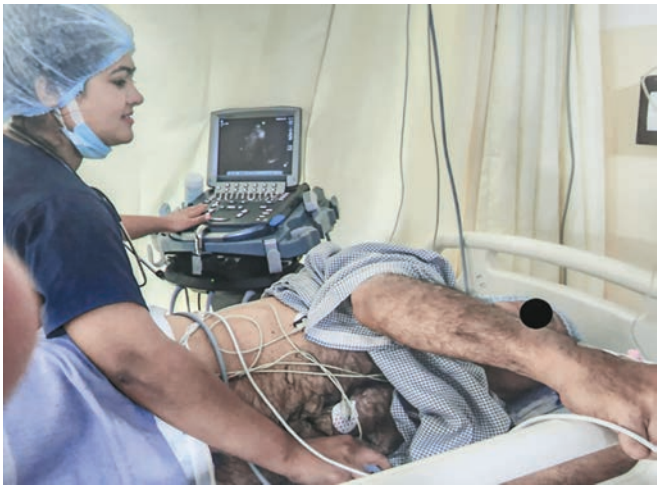


Fig. 14: Performing two-dimensional echocardiogram and tele-intensivist assessing through camera.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

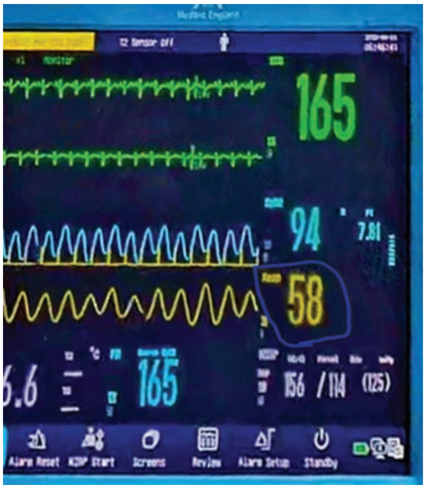


Fig. 15: Monitor showing tachypnea.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

Respiratory System

- *Tachypnea/bradypnea:* This is a common finding in ICUs, which can be picked up on camera **(Fig. 15)**. Work of breathing often helps the tele-ICU physician to understand and preemptively plan for the worsening condition of a critically ill patient. The bedside RMP/ primary consultant can prepare for intubation and mechanical ventilation, counseling the family member regarding the same **(Fig. 16)**. This can often result in a planned intubation as opposed to a crash intubation avoiding the risks that come with it.
- *Desaturation:* Desaturation can often be accompanied with bradypnea or tachypnea with respiratory distress. These events can be picked up by remote services and are informed to the bedside team as well as the telephysician.
- *Respiratory distress management:* The need for noninvasive ventilation (NIV)/invasive ventilation is assessed with the help of RMP's examination findings. If required, necessary information regarding type of the mask, how to fix the mask, prevention of mask leak, and ventilator settings **(Fig. 17)** are communicated. The remote team can even train the bedside caregiver **(Figs. 18 and 19)** regarding the application of the mask and supervise an intubation remotely.

Gastrointestinal System

- *Gastrointestinal bleed:* After collecting the history of the patient from RMP/bedside nursing staff, telephysicians can assess the need for blood transfusion and suggest other specialty references (gastroenterology). Telephysicians may also suggest the need for endoscopy. Ryle's tube aspirate can be reviewed remotely, which may help with decision-making.

Fig. 16: Bedside practitioner examining the patient.
Source: Care Centre, Cloudphysician Healthcare: Captured from real-time camera with permission.

- *Abdominal distension:* Abdominal distension can be seen through PTZ camera. With the help of cameras, certain procedures can be monitored and step-by-step advice can be given in real-time minimizing the risk of adverse events **(Fig. 20)**.

Renal System

- *Anuria/oliguria/pyuria:* Remote care can monitor urine output and ensure that patients do not get volume overloaded. Telephysicians can also help in dosing the renal replacement therapy (RRT) sessions based on ABGs.
- *Hematuria:* Presence of hematuria can be easily detected by the video system. They can also identify and guide what next to be done for the same. For instance, stopping prophylactic heparin and follow-up of hemoglobin levels can be done by the remote expert assisting the bedside team.

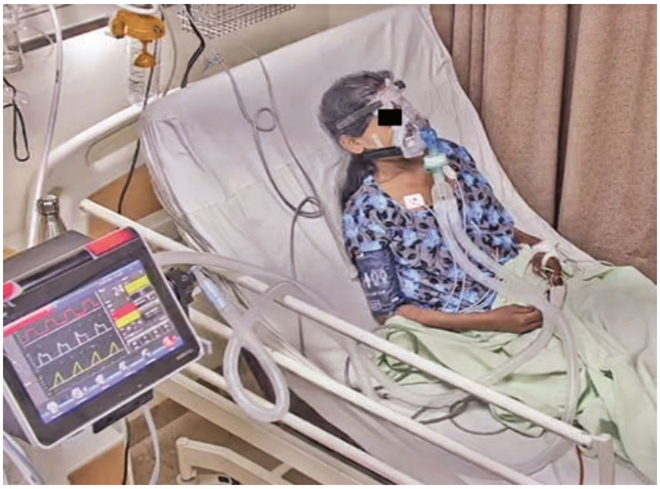


Fig. 17: Ventilator graphs and patient stability on noninvasive ventilation (NIV) can be accessed remotely.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

Trauma

Primary approach in managing trauma patients is the initial assessment by visualizing the patient. PTZ cameras can help in visualizing the patient and assess the general condition. Few external organs that can be assessed via PTZ camera are as follows:

- *Scalp:* Cut lacerated wound and degloving injury of scalp
- *Eyes:* Racoon eyes, penetrating injury to eyes, and pupillary reactions
- *Nose, mouth, and ears:* Nasal bleed and cut lacerated wound around nose, mouth, and ear
- *Chest:* Superficial injuries on the chest, penetrating injuries of chest, and chest movements during respiration
- *Abdomen:* Penetrating injuries of abdomen and abdominal distension
- *Limbs:* Superficial injuries of the limbs and swelling
- *Genital organs:* Superficial injuries of the genital organs

CASE SCENARIO 1

A 24-year-old male patient presented with an alleged history of a road traffic accident. The patient was a motorcycle rider who had hit an electric pole while on the motorcycle. On

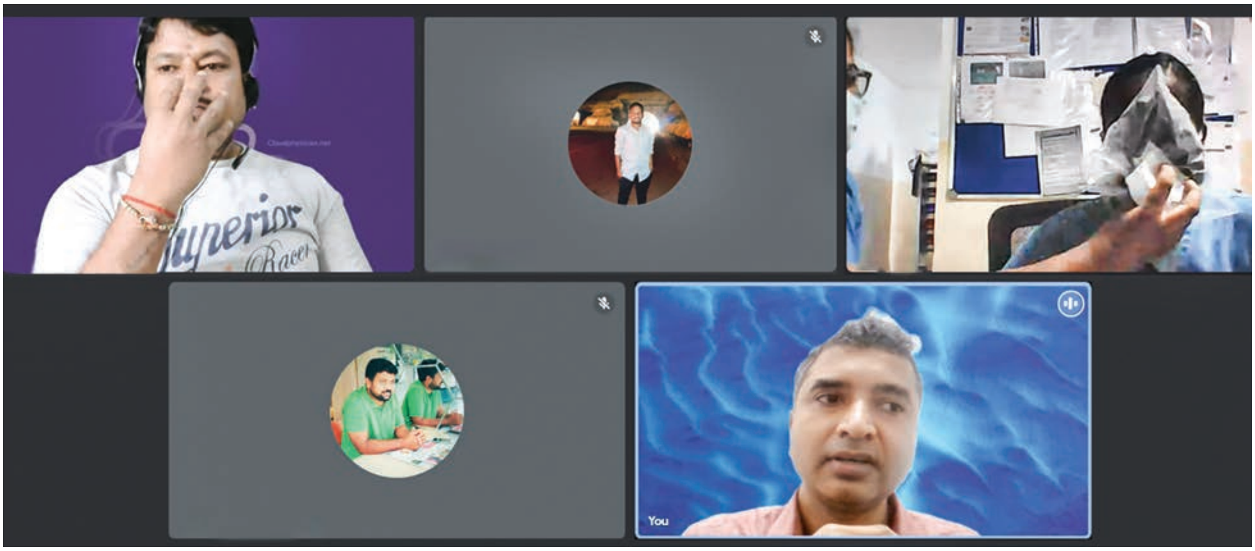


Fig. 18: Training of NIV mask application for bedside nursing staff remotely.
Source: Care Centre, Cloudphysician Healthcare; A real-time teleconversation.

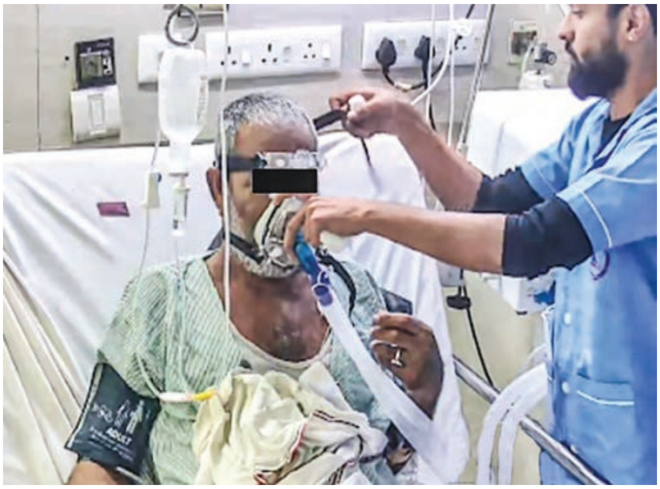

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Fig. 19: Nursing staff connecting the NIV after discussing it with the remote providers.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

[redacted]

Fig. 20: Monitored feeding to ensure safety in critically ill patients.
Source: Care Centre, Cloudphysician Healthcare; Captured from real-time camera with permission.

arrival at the emergency room, the duty doctor examined the patient and informed the tele-ICU consultant (as per the protocol).

General Examination

The patient was conscious, oriented, moving all four limbs, hemodynamically stable, and maintaining saturations on minimal O_2 supplementation. He had sustained an injury to the head, face, wrist, and his right eye was ecchymotic. The tele-ICU consultant reviews the examination findings of the bedside doctor assisted by high-definition cameras. Furthermore, the case is discussed with the duty doctor in real time focusing on the management plan, which includes immediate treatment options and laboratory and radiological investigations.

Pertinent history that can be taken remotely:

- Does the patient have any pain in any other part of the body?
- What was the speed during the accident?
- Did he have any loss of consciousness, vomiting or seizures?

Answer 1: Patient had pain in the left wrist. An X-ray of the left wrist with the forearm was ordered to rule out any fractures.

Answer 2: The speed was approximately around 40–50 km/h. The speed of the vehicle is relevant to distinguish the type of injuries possible.

Answer 3: There was no history of loss of consciousness, vomiting, or seizures. All these will suggest a possibility of raised intracranial pressures.

Investigations

In addition to routine laboratory tests, creatinine phosphokinase (CPK) has significant relevance, which helps in estimating the extent of muscle injury and liver function tests can help assess the possibility of liver damage.

In an emergency, an E-FAST (Extended Focused Assessment with Sonography in Trauma) scan can be instrumental in assessing pathology in the abdomen and the thorax, which in turn helps in making the decision of early surgical interventions if required such as exploratory laparotomy, pleural or ascitic tapping. Furthermore, a whole body CT scan is warranted if the patient is relatively stable. This helps us in ruling out any intracranial, intra-abdominal, and thorax pathologies with a greater degree of confidence. Tele-ICU consultants can guide the duty doctor in performing the E-FAST scan and can eventually visualize the ultrasound screen via camera.

In this case, a CT scan was performed and was suggestive of pneumomediastinum around the pericardium, great vessels, and esophagus. A CT brain was suggestive of pneumocephalus, frontal bone fracture, base of skull fracture, and contusions. A CT scan of the face suggested fractures of maxilla and the mandible. The CT of the spine was normal as was the CT of the abdomen.

Treatment

The patient was initiated on antibiotics and other supportive treatments as per the tele-ICU consultant's advice. Neurosurgical consultation was taken and the plan for conservative management with 100% of O_2 to reduce pneumocephalus was followed. The case was discussed with the neurosurgeon and maxillofacial surgeon by the teleconsultant.

The patient's condition was assessed by a teleconsultant during rounds with the bedside duty doctor.

The patient underwent surgery for facial bone fixations by a maxillofacial surgeon. Supportive care, which included physiotherapy and feeding, was continued. The patient was being monitored by the remote monitoring services 24×7.

The patient's condition improved and he was subsequently discharged.

CASE SCENARIO 2

A 75-year-old male patient presented with breathlessness, abdominal pain, and inability to pass urine and stool since morning.

General Examination

The duty doctor takes an appropriate history and does a head-to-toe examination of the patient. As per the history and chief complaints, organ systems that are likely involved are the abdomen and the respiratory system.

The patient is tachypneic and maintains saturations on O_2 supplements. His blood pressures are borderline with mean arterial pressures around 65 mm Hg.

Systemic Examination

On examination of the respiratory system, the duty doctor found that the patient had bilateral crepitations and wheezing on auscultation. The examination of the abdomen was done and it was reported to be tense, tender, and distended.

History of Presenting Illness

After discussing with the teleconsultant, a few leading enquiries were made by the duty doctor with respect to the respiratory system, such as:

- Does he have a history of respiratory illness?
- Does he have a history of COPD/hyperactive airway disease?
- What was his occupation?
- Does he consume any medications?

Answer 1: He had a history of interstitial lung diseases diagnosed 2 months ago. He has a high-resolution CT (HRCT) report suggesting the same.

Answer 2: He did not have COPD or a reactive airway disease.

Answer 3: He was a government employee and his occupation does not expose him to allergens or other toxins that may cause lung disease.

Answer 4: He was on steroids and antifibrotics for a few months for interstitial lung disease.

Investigation

Relevant investigations that include serum amylase and lipase to rule out pancreatitis, serum lactates for gut ischemia, and an ABG to rule out any acid–base disorder were done. Radiological investigations including chest X-ray or a HRCT, ultrasound, or a CT abdomen in this case would be appropriate. The chest X-ray was suggestive of increased fibrotic markings and a CT abdomen suggested a possibility of perforative peritonitis.

Treatment

The teleconsultant suggested the plan of action required after reviewing all the laboratory and radiological reports. The primary consultant was informed about the urgency of laparotomy and a surgical consultation was given. The teleconsultant guided the duty doctor in making the necessary arrangements for emergency surgery. The teleconsultant also counseled the patient's family regarding the severity of the condition and need for emergency surgery. The duty doctor made all the arrangements as suggested by the teleconsultant and the patient was taken up for surgery as soon as the surgeon arrived.

The patient was shifted to ICU after the laparotomy and closure of the duodenal perforation for further management. The teleconsultant then augmented the ICU care by continuous monitoring and making an appropriate plan of action on a daily basis. As an example, the patient had hypotension, which was picked up by remote monitoring services and an actionable alert to the bedside team enhanced the quality of care. The possibility of sepsis was discussed with the surgeon and antibiotics were escalated after collecting blood, urine, and endotracheal secretion for culture and sensitivity. The teleconsultant guided the duty doctor in performing echocardiography and assessed the hemodynamic status of the patient via high-definition cameras. Appropriate management plan of administering fluid bolus and later initiating vasopressors was also advised to the doctor. Daily laboratory investigations were monitored and acted upon based on the abnormalities. Eventually the patient was assessed for respiratory weaning and weaning of vasopressors. Eventually, the patient's condition improved and the patient was shifted to a ward.

FAST HUG BID

The ICU bundles help streamline and standardize care. Remote teams can work with the bedside nursing team and help assist the primary provider to carry out these workflows. For example, FAST HUG BID is a commonly used bundle that comprises feeding, analgesia, spontaneous breathing trial, temperature, head position, ulcer prophylaxis, glycemic control, bowel movements, indwelling catheters, and drug review. Remote teams can ensure that these components of care are protocolized and can reduce workload on the bedside team. A dual check often can increase compliance and enhance outcomes related to these bundles.

F: Feeding is critical to expedite recovery of an ICU patient. Telenutritionists can ensure the patients get an appropriate amount of proteins, carbohydrates, and fats after looking through the case files and discussing with bedside healthcare workers. Telenutritionists can manage enteral feed intolerance, if any, in critically ill patients and improve overall outcomes. RPM services are also able to monitor RT position, aspirate, and keep a track of frequency of feeds. Telenutritionists can also advise regarding total parenteral nutrition (TPN) and peripheral parenteral nutrition (PPN) and titrate delivery.

A: Analgesia can be remotely monitored pain scale scoring via camera visualization is easy to do. Telephysicians advise regarding the analgesic medications and prevent harm by monitoring dosing and analgesia response. This in turn helps improve outcomes and reduce ICU expenses.

S: Spontaneous breathing trial (SBT) is an important aspect in the care of a ventilated patient. Daily spontaneous breathing trial reduces patient ventilatory days, ventilator-associated pneumonia (VAP), and ICU stay. Telephysicians can assess the patient for readiness for extubation by SBT and can assist the bedside team in extubating the patient. Their presence can also ensure that the SBT is carried out per protocol.

T: Normothermia helps organs of the body function at optimal levels. Hyperthermia/hypothermia both hamper the physiological functioning of the body and have detrimental outcomes. RPM services can monitor the temperature and can alert the bedside regarding maintenance of normothermia and trigger laboratory tests that may be required to evaluate the cause of abnormalities.

H: Head-up position helps in reducing aspiration. A head-up position of 30–45° prevents VAP and increases oxygenation by preventing atelectasis.

U: Recently the need for stress ulcer prophylaxis has been deprioritized due to new evidence that perhaps their utility may be restricted to a subset of critically ill patients. The remote team can ensure that only patients with valid indications get prophylaxis, thus minimizing the use of these drugs and potentially reducing harm and cost associated with their administration.

G: Maintaining normoglycemia in a critical patient reduces the chance of infection and length of ICU stay. Remote teams often have the ability to assist with insulin protocols and their adherence by the bedside.

B: Bowel movements must be monitored in all ICU patients and telephysicians can guide and review medications that may contribute to abnormal bowel movements. Diarrhea may signal the onset of life-threatening conditions such as a *Clostridium difficile* infection that can readily be addressed by remote teams thus saving time.

I: Indwelling catheters are a source of infection and reducing their use can decrease hospital-acquired infections. Daily reviews by teams, both bedside and remote, can ensure that catheter sites are healthy and do not show signs of infection. Protocolized strategies to remove them daily can be enhanced with a remote team in place.

D: Drug reconciliation helps ensure that ICU patients are on the lowest number of drugs possible and thus reduces the chance of errors such as duplication or over and underdosing. Such methodical reviews can also help with drug interactions and antibiotic de-escalation. Reviews of the electronic record can be done by the remote team for many patients in a network saving time and ensuring adherence to protocols.

FAMILY COUNSELING

Engaging the family of a critically ill patient is fundamental to ensuring that they are involved in the care of their loved one and that they are empowered to assist in the complex decision making that is often seen in modern ICUs. The bedside teams can utilize the remote providers to reinforce their communication and messaging to family members. Often such repetition helps build trust and solidify the plan of care for family members. The tele-ICU providers can often act as a first responder for these instances as their availability can be ensured in minutes. This again helps in building trust. It is critical for the family to understand that the remote providers are not a replacement but are augmenting the bedside team. The communication can also be recorded for future reference. Remote services may even bridge the gap between the primary physician and the patient at the time of discharge to ensure safe continuity of care. Consent and privacy are important to consider, and this space is dynamic in India. We urge the readers to review this elsewhere as it is out of the scope of the current review.

REGULATORY CONSIDERATIONS OF TELE-INTENSIVE CARE UNITS

Until recently, guidelines have not been explicitly clear on how to practice telemedicine in India. In 2020, telemedicine practice guidelines were published. They have since been ratified as a gazette notification as well and can be found in the link https://www.mohfw.gov.in/pdf/Telemedicine.pdf. Certain acts and rules govern the practice of medicine and information technology. These acts include:

1. Indian Medical Council Act, 1956
2. Indian Medical Council, Drugs & Cosmetics Act, 1940 and Rules 1945
3. Clinical Establishment (Registration and Regulation) Act, 2010
4. Information Technology Act, 2000
5. Information Technology Rules 2011

There are still gaps in the framework that are likely to be addressed by upcoming legislation such as the Personal Data and Protection Bill.

Need for Guidelines

Future guidelines will need to address important issues such as:

- Physician–patient relationship protocols
- Guideline for liability and negligence in remote care
- Informed consent
- Continuity of care
- Medical records privacy and maintenance
- Patient record privacy and security
- Prescribing treatment for both controlled substances and advising procedures remotely
- Health education and counseling
- Role of social media and prevention of its use in formal healthcare.

FUTURE DIRECTIONS

Clinicians are already using technology in various forms today in the management of their patients. A few examples are teleconsultations, clinical decision support systems (CDSS), and even risky use of social media. A multidisciplinary approach in management of patients is often required while managing a patient and future health record platforms will likely be more collaborative in nature. These will help doctors in making decisions, enable them to work from any location, and provide care to patients across the globe. Solutions designed in resource-constrained settings have the potential to shape care for other parts of the world that are facing similar problems.

The use of technologies such as artificial intelligence (AI) and Big Data will help healthcare workers in making early decisions that are data driven and benefit from the latest developments in science, making research agile and relevant as soon as possible. Evolution of these operational and technological methods will help bridge care gaps and allow clinicians to take care of more patients than ever before with better quality and fewer errors.

SUMMARY

This chapter illustrates the impact of tele-ICU technology and service on ICUs in remote areas, detailing the necessary infrastructure, technology, and protocols for effective operation. It covers key aspects such as hardware, software, networking, staffing roles (including remote specialists and bedside staff), operational workflows (patient monitoring and proactive response), and integration with hospital electronic systems (EMR, EHR, and data security). The chapter also discusses positive outcomes, challenges of implementation, cost-effectiveness, and mortality reduction, highlighting successful case studies from hospitals that have integrated tele-ICU services.

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Chapter 40

How will I Approach/Examine a Patient with an Alleged Hanging?

Radha Moda Gururaja Rao

ABSTRACT

Hanging is one of the most common way of suicide. Self-harm with hanging is termed near hanging and manifests with multiorgan dysfunction predominantly cardiorespiratory and neurological. In the absence of specific guidelines for hanging, approach is in the lines of advanced trauma life support (ATLS) format. Survival in this group of patients is optimistic and hence resuscitation and stabilization are advocated and have to be sustained.

INTRODUCTION

India reports a higher number of suicide deaths. Suicide accounts for more deaths in the age group of 15–29 years compared with other causes of death. Hanging is the most common method of suicide, preceded by pesticide poisoning, followed by medicine overdose, and self-immolation. Several risk factors such as depression, alcohol abuse, and social and cultural aspects have been identified as risk factors predisposing to suicide. Hanging is more common due to the easy availability of ligature material, painless, bloodless, and quick death.

Suicidal hanging is defined as a form of asphyxia death produced by the body being self-suspended by a ligature material around the neck, with the body's weight serving as the compression force around the neck. Usually, rope or readily accessible clothing serves as the ligature material. The hanging may cause death by various processes that function separately or in combination. These include spinal cord injury, venous and arterial blockage, airway obstruction, and carotid complex compression, resulting in reflex cardiac arrest, and so on.

Hanging poses many challenges for a clinician's bedside, as the majority of the time it presents with multiorgan involvement. Clinicians, emergency physicians, and intensivists should be knowledgeable and aware of the possible sequelae and meticulous while managing these sets of patients in the intensive care unit (ICU).

CASE SCENARIO

A young taxi driver after leaving kids at school returns home on a regular day and is supposed to relax after his night shift. The wife leaves home for her job. In the next 15 minutes, the wife gets a call that her husband was found hanging at the residence and not responsive. He was rushed to the hospital; it was an out-of-hospital cardiac arrest, and he was resuscitated in the emergency room after the return of spontaneous circulation he was moved to the medical ICU for postresuscitation care. Clinically he was in a coma with Glasgow Coma Scale (GCS) –2 + T. He was commenced on controlled mechanical ventilation, neuroprotective measures, and standard of care in ICU like enteral feeding deep vein thrombosis prophylaxis.

Diagnosis in hanging is not in question the majority of times, but the workup on the other hand is not straightforward.

Possible substance abuse, alcohol intoxication, or a combination of other methods of self-harm with hanging, should always be kept in mind. At the same time, we take history and actively search for manifestations too. The major *pathogenic mechanism of death is vascular rather than asphyxia*.

The jugular venous system is superficial and most vulnerable, and with the pressure of 2 kg, occlusion occurs,

TABLE 1: The difference between judicial and nonjudicial hanging.

	Judicial	*Nonjudicial*
Ligature	Below the chin	Below the occiput
Suspension	Complete	Incomplete
Sequelae	High cervical fracture cord transection	<5% cervical fracture

and resultant stagnant hypoxia causes cerebral hypoxia and edema.

Carotid artery occlusion occurs at a pressure of 5 kg. Tracheal compression at 15 kg and vertebral artery compression at 30 kg.

It is important to understand the difference between judicial and nonjudicial hanging as pathophysiology is different **(Table 1)**.

DEFINITIONS

- "Complete hanging" is defined as when a patient's legs are fully suspended off the ground and the patient's body weight is fully suspended by the neck.
- Incomplete hanging" is defined as when some part of the patient's body is still on the ground and the body's full weight is not suspended off the ground.

Complications and clinical courses follow depending on the injury to neck structures and time of medical attention. Local and systemic complications are mentioned in **Figure 1**.

In the absence of a dedicated guideline for the management of near-hanging patients, the approach largely described in the literature is along lines recommended by the advanced trauma life support guidelines, primarily including immobilization of the neck, securing airway by endotracheal intubation, positive-pressure ventilation, maintenance of fluid, electrolyte balance, euglycemia and normocarbia (to control intracranial tension).

INITIAL APPROACH

- Approach according to, stratifying risk based on systemic injuries
- As a clinician, the approach and evaluation proceed with stratifying risk to neurologic, vascular, respiratory, and deep musculoskeletal structures.
- Initial stabilization of airway, breathing, and circulation (ABC) as always, and subsequent continued monitoring for delayed sequelae would be done.

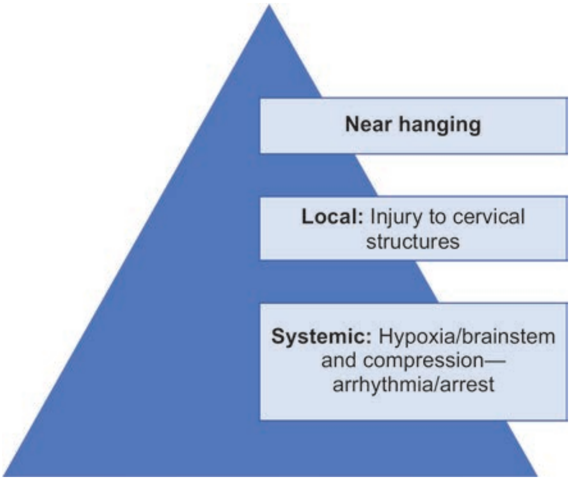

Near hanging
Local: Injury to cervical structures
Systemic: Hypoxia/brainstem and compression—arrhythmia/arrest

Fig. 1: Local and systemic sequelae in near hanging.

- Targeted testing is adequate in low-risk patients but needs liberal diagnostic workup in patients in high risk and needing respiratory support with neurologic impairment.
- Low risk are those who have been brought with alleged hanging are clinically sane with no evident airway obstruction and do not have hoarseness/dysphonia.

Initial Stabilization

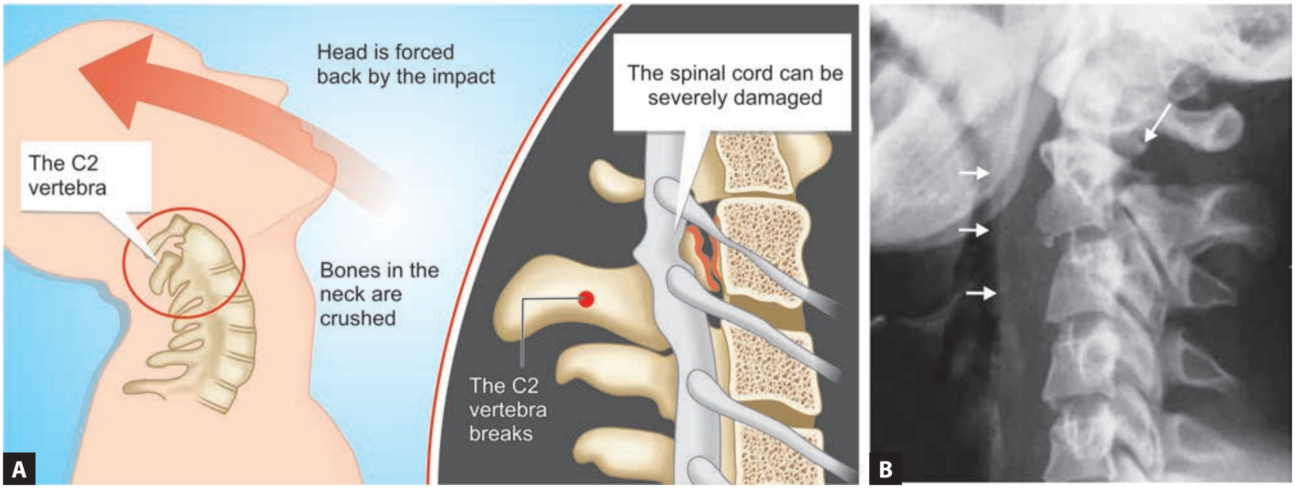
- Airway with cervical spine precautions
- Breathing
- Circulation
- Disability assessment

Airway

Inspect carefully for stridor/dysphonia/respiratory distress, airway obstruction, muffled voice, or altered mentation. Anticipate a difficult airway—cervical spine trauma and airway edema, both can predispose to a difficult airway. Prepare for a difficult airway and be ready for a surgical airway.

Classical teaching is that cervical fracture—a hangman's fracture seen in judicial hanging in which the knot is below the chin, would hyperextend the C2 vertebra and result in spinal cord transection **(Figs. 2A and B)**. The constriction force is the entire body weight, the drop is more than the height of the patient, and the resultant effect is cervical spine fracture.

Cervical spine fracture is mechanistically not achievable in self-hanging and <5% have a cervical injury. Los Angeles County study (Hua A et al.,) advocates a low


Head is forced back by the impact
The C2 vertebra
Bones in the neck are crushed
The spinal cord can be severely damaged
The C2 vertebra breaks
A
B

Figs. 2A and B: Emergency medicine education—managing hanging injury.
Courtesy: emDOCs.net.

threshold for cervical spine imaging, though the incidence of spine fracture in nonjudicial hanging is less. Cervical spine precautions must be taken.

Breathing

The patients can have a cough, stridor, dysphonia/muffled voice, or aphonia. Audible crackles if pulmonary edema has set in. Call for help. Airway compromise can be rapid/life threatening/delayed. Keep ready a video laryngoscope, bougie, or a difficult airway cart. Early airway protection with little warning and aggressive airway management in the presence of unconsciousness/dyspnea/hoarseness. If endotracheal intubation is unsuccessful, cricothyroidotomy/translaryngeal ventilation should be considered.

Airway assessment with ultrasonography will help with distorted anatomy.

Circulation

Arrhythmia and shock are common and need to be looked for. Judicious and cautious fluid resuscitation is needed as overzealous fluid would aggravate acute respiratory distress syndrome (ARDS) and cerebral edema.

Disability

Depending on the extent of hypoxic injury, GCS would be variable, and hypoxic injury postcardiac arrest could worsen the brain injury. Encephalopathy is multifactorial—hypoxia, intoxication before self-hanging, and cerebral edema. Neuroprotective measures to be adopted. Avoid secondary insults such as hypoxia hypercarbia, hyperglycemia, and hypotension which aggravates brain injury **(Fig. 3)**.

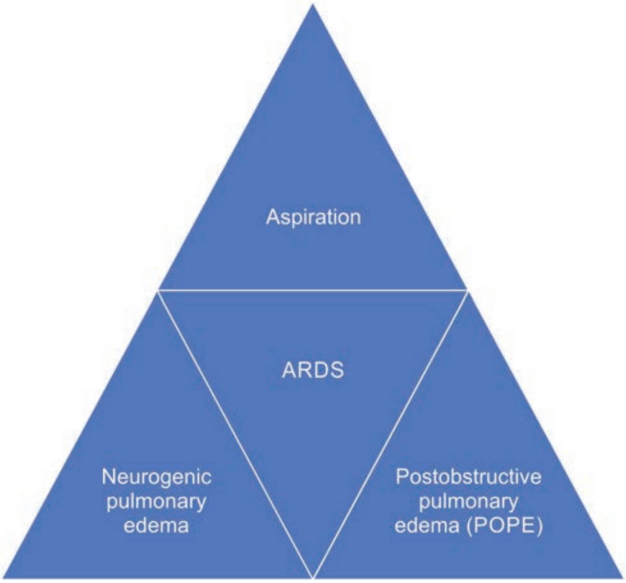

Aspiration
ARDS
Neurogenic pulmonary edema
Postobstructive pulmonary edema (POPE)

Fig. 3: The development of acute respiratory distress syndrome (ARDS) is multifactorial.

GENERAL PHYSICAL EXAMINATION

- *Head and face:* Periorbital edema, swelling of the conjunctiva, subconjunctival hemorrhage, congestion of face, petechiae–mucus membranes, skin, tongue edema, and cyanosis (lips).

- *Neck:* Ligature marks, ecchymosis, hematoma at neck, and crepitus.
- *Trunk, perineum, and extremities:* Subcutaneous emphysema in lower neck and upper chest urinary incontinence, abrasions, and injury marks in feet and leg in case of partial hanging.

SPECIFIC EXAMINATION

Local Examination of the Neck

- *Inspect for external wounds:* Abrasions, ecchymosis/laceration around the neck, and also for ligature marks and extent of ligature marks.
- Painful tongue movements could be due to epiglottis or hyoid bone injury.
- *Tardieu marks:* Petechial hemorrhages on the conjunctiva, mucous membranes, and skin cephalad to ligature, are due to mechanical obstruction to the venous flow.
- Palpate for crepitus and step off along the cervical spine anteriorly to rule out fracture.
- Palpate for carotid bruit.

Further Management

- Expect early pulmonary complications.
- Patients with symptoms of laryngeal or tracheal injury (e.g., dyspnea, dysphonia, aphonia, or odynophagia), should undergo laryngobronchoscopy by the ear, nose, and throat (ENT) department.
- When suicide is suspected, evaluate patients for other methods of self-harm (e.g., wrist lacerations, self-stabbing, and ingestions). It is also important to consider drug and alcohol intoxication.

SYSTEMIC EXAMINATION

Respiratory

- Look for tachypnea, active use of accessory muscles of respiration, inability to complete sentences, and hypoxia.
- Variable dynamic respiratory failure is to be anticipated.

Neurogenic pulmonary edema can happen early, as early as 4 hours, and as late as 12–24 hours into hanging. Neurogenic pulmonary edema resolves in 72 hours.

Lung ultrasound may show multiple B lines—reverberation artifacts that are hyperechoic and signify interstitial edema, which if more than three are pathological **(Fig. 4)**.

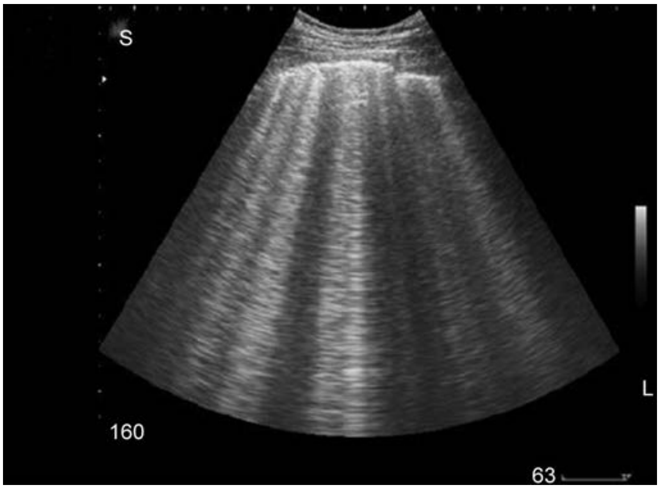


Fig. 4: B-lines on lung ultrasound.

Cardiac

- Look for arrhythmias—secondary to vagal injury, which can result in reflex bradycardia or sinus arrest can also happen.
- Shock is common and is secondary to neurogenic causes.
- Dysautonomia
- Takotsubo cardiomyopathy—reversible heart failure, secondary to release of catecholamines, uncommonly seen in hanging.

Neurological

Variable encephalopathy depends on the degree of hypoxic insult and occurrence of cardiac arrest. GCS assessment is to be done meticulously with pupillary charting.

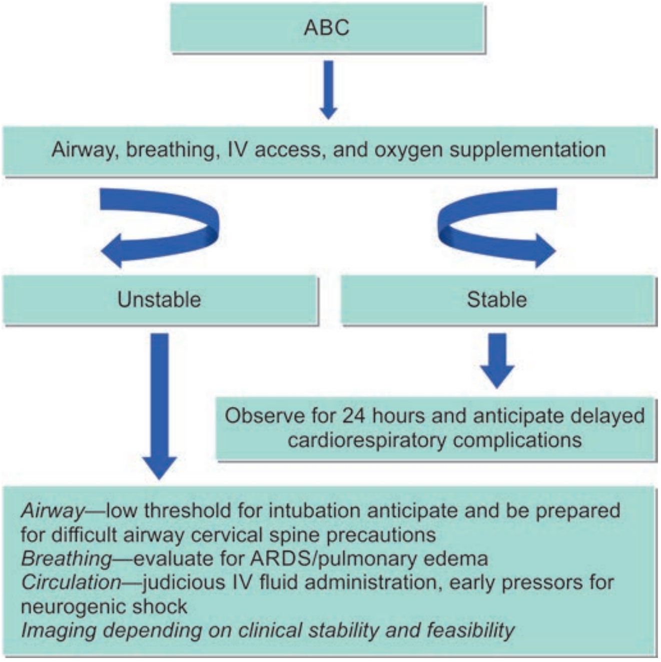
Patients can have status epilepticus secondary to hypoxic brain injury. Which will need to be managed appropriately with antiepileptic drugs.

Algorithmic approach after ABCD assessment represents in **Flowchart 1**.

IMAGING/FURTHER TESTING

- Chest X-ray—to look for aspiration, ARDS, or pulmonary edema.
- X-ray neck—hyoid fracture and surgical emphysema.
- Computed tomography (CT) scan of the cervical spine to look for fracture/dislocation of the vertebrae.
- CT of the brain in unconscious patient to look for cerebral edema/hypoxic—anoxic injury.

Flowchart 1: Algorithm for alleged hanging patients.


ABC
Airway, breathing, IV access, and oxygen supplementation
Unstable
Stable
Observe for 24 hours and anticipate delayed cardiorespiratory complications
Airway—low threshold for intubation anticipate and be prepared for difficult airway cervical spine precautions
Breathing—evaluate for ARDS/pulmonary edema
Circulation—judicious IV fluid administration, early pressors for neurogenic shock
Imaging depending on clinical stability and feasibility

(ARDS: acute respiratory distress syndrome; IV: intravenous)

- CT angiography if focal neurological deficits are present. Magnetic resonance imaging (MRI) brain may be necessary to assess the severity of hypoxic brain injury.
- Imaging should be done according to the clinical feasibility and stability of the patient.
- Toxicology screening when in doubt to be done.

PROGNOSTICATION

While the overall survival rates described in patients with near hanging are optimistic and range from 70 to 100%, factors predicting clinical outcomes have been variably described and remain largely inconsistent. The most controversial of them is the GCS score. However, even with GCS <3, in a case series, up to 32% survival has been documented, emphasizing the need for aggressive initial resuscitation.

POOR PREDICTORS

- GCS <3 has a poor outcome
- Hanging time >5 minutes
- Cardiac arrest on arrival
- Cervical spine injury

DISPOSITION

- Even if the initial presentation is clinically benign, all near-hanging victims should be observed for 24 hours, given the high risk of delayed neurologic, airway, and pulmonary complications.
- Psychiatry consultation is vital before discharge from the hospital.
- Emphasize strict return precautions and education about possible delayed respiratory and neurologic dysfunction when discharging patients' home.

SUMMARY

Hanging poses many challenges to clinicians. Organ specific supportive care is vital. Possible other ways of self-harm like substance abuse (e.g. use of alcohol) should be kept in mind and accordingly evaluated. Varied multiorgan manifestations predominantly due to neurovascular and airway injury in the neck pose challenges bedside for clinicians and needs meticulous evaluation and management.

SUGGESTED READING

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Chapter

41

How will I Approach/Examine a Patient with Skin and Soft Tissue Lesions?

Dipali Anand Taggarsi, Natesh Prabu R

ABSTRACT

Any patient evaluation will be incomplete without a comprehensive evaluation of the patient's skin. While in some instances, the primary disease may be of the skin and soft tissue, in others, skin lesions may help lead to a conclusive diagnosis.

In this chapter, we will be discussing two aspects of the examination of the skin and soft tissue. One segment will focus on the examination of skin lesions, cutaneous manifestations of systemic diseases, and rashes in an intensive care unit (ICU). The second will be an approach to skin and soft tissue infections.

PART I: EXAMINATION OF SKIN AND SOFT TISSUE INFECTION (SKIN)

INTRODUCTION

Dermatological manifestations in the ICU are commonly overlooked during an initial assessment. They can be present during the admission and may or may not be related to the illness. They may also develop during the course of the ICU stay and may result from various factors such as inadequate tissue perfusion, body temperature changes, prolonged immobilization, multiple drug use, invasive procedures, etc.

Several critically ill patients present with varied types of dermatological manifestations to the ICU. These may be divided as:

- A primary disease of the skin resulting in the critically ill state, for example, Stevens–Johnson syndrome (SJS) or erythema multiforme
- Skin disease unrelated to the critically ill state, for example, skin changes seen with chronic venous congestion
- Dermatological manifestations of the underlying medical condition that may provide diagnostic value [e.g., characteristic rashes in systemic lupus erythematosus (SLE), palpable purpura in infective endocarditis, hyperpigmentation in adrenocortical insufficiency, or eschar in rickettsial disease]
- Signs of complications that have arisen due to the primary disease such as mottling of skin or purpura fulminans in septic shock, petechial rashes secondary to thrombocytopenia, or pressure sores following prolonged immobilization
- A result of complications of therapy—drug rashes, thrombophlebitis, pressure sores, device-related injuries, and secondary to vasopressor drug use.

When patients present with abnormal skin conditions, a detailed history of the condition and examination must be done.

CASE SCENARIO

A 25-year-old female presented with history of fever and cough for 3 days. She visited a local hospital where she was given a prescription of amoxicillin clavulanate. After taking two doses, she noticed redness over her chest associated with itching. She presented to the emergency department with worsening cough, wheeze, and breathing difficulty. On examination, she was found to be tachycardic and hypotensive and had bilateral rhonchi. Examination of her rash revealed it was erythematous and blanching.

History and Symptomatology

A great deal of attention to dermatological manifestations should be paid while taking history as well as examining patients. In some instances, patients or families notice subtle changes and document them with photographs, which may provide important diagnostic clues. In other instances, the skin changes may go unnoticed by the patient and family.

Whenever a history of skin lesions is given, it is important to note their onset and progression that may have occurred with time. Lesions may start with redness or swelling, and patterns vary. Timing of the lesion may give a clue as to whether it is a primary skin disease, related to consumption of food or a drug, or part of a constellation of symptoms of the underlying disease. Some rashes such as urticarial rashes may be transient.

History of any associated itching, pain, or discharge from the rash may provide diagnostic clues **(Fig. 1)**. Pain preceding the appearance of skin changes may indicate an acute vascular pathology such as arterial occlusion or pain preceding redness and swelling can be seen in necrotizing fasciitis (NF). Herpes zoster rashes are also extremely painful. Drug-induced allergic rashes can be associated with pruritis. Loss of sensation in the affected area may indicate Hansen disease or sometimes the presence of sensory motor neuropathy, as in certain types of vasculitic syndromes.

Associated systemic symptoms give a clue to the severity of rash. The presence of fever, low blood pressure, which may be manifested in the form of excessive fatigue, giddiness, postural hypotension, and reduced urine output indicate that there may be multisystem involvement requiring urgent attention.

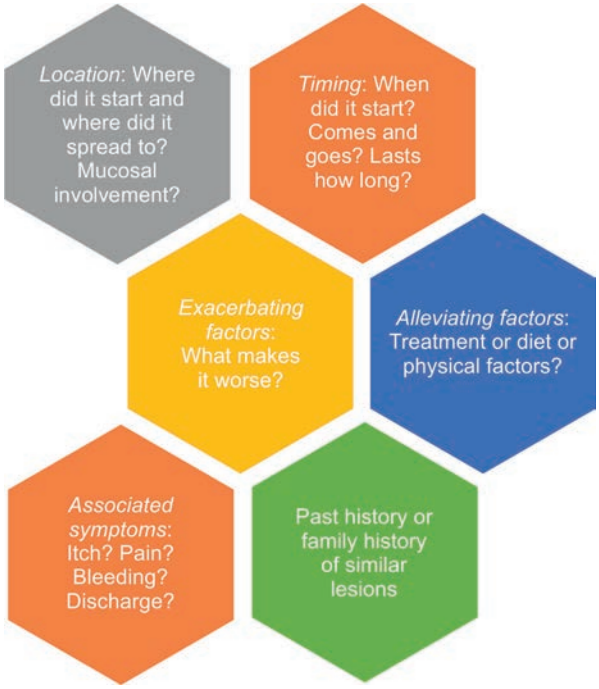

Location: Where did it start and where did it spread to? Mucosal involvement?
Timing: When did it start? Comes and goes? Lasts how long?
Exacerbating factors: What makes it worse?
Alleviating factors: Treatment or diet or physical factors?
Associated symptoms: Itch? Pain? Bleeding? Discharge?
Past history or family history of similar lesions

Fig. 1: History-taking for rashes in an intensive care unit (ICU).

History of all types of food, drug, and other allergies must be elicited. History of mucosal involvement can be elicited by asking for a history of burning in the oral cavity, presence of oral or genital ulcers, pain while swallowing or during urination, which may be seen in erythema multiforme major. The presence of associated symptoms such as joint pains, hair loss, fatigue, and oral ulcers may indicate the presence of an underlying autoimmune disease.

Relevant history that may point toward an etiology will also include history of travel (tropical fevers such as rickettsial disease, leptospirosis, malaria, and dengue may come with travel history), history of snake bite or insect bites, and history of recent trauma.

General Physical Examination of Skin

Examination of skin requires complete exposure after taking patient consent, if the patient is awake. Adequate lighting should be ensured. Permission should be taken from the bedside nurse. Screens should be used to ensure privacy of the patient. Proper hand hygiene should be performed before touching patients or their surroundings.

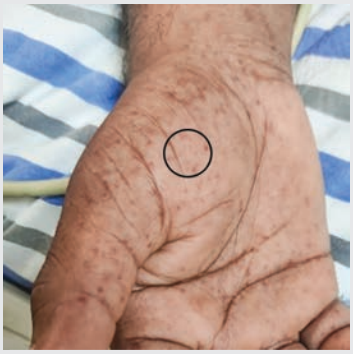
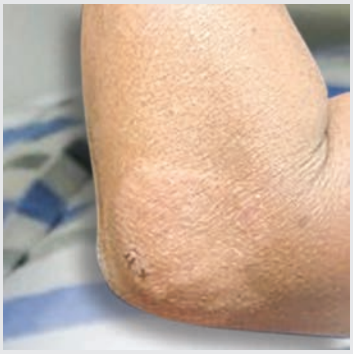
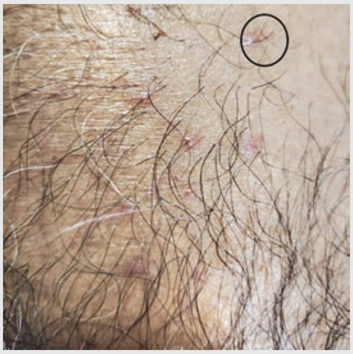
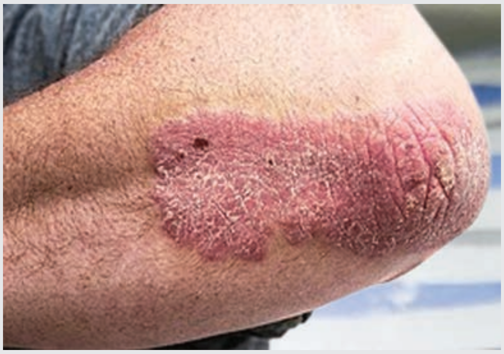
Inspection

The initial assessment of skin while first examining a patient should include inspection for any erythema, papules, macules purpuric patches, vesicles, bullae, abnormal scaling, etc. The site and size of these lesions must be documented in the chart. **Table 1** describes some of these lesions and is accompanied by images. A thorough inspection includes examination of axilla, back, abdomen, palms and soles, mucous membranes, hair, and nails.

This skin examination is not limited to initial assessment alone. It should be done daily for all patients and sometimes even several times during the day based on progress of the disease state. Development of new skin lesions may be a new symptom of a hitherto undiagnosed condition, allergic reaction, pressure sore formation, or a new condition altogether.

Skin examination in trauma becomes important. Examination and documentation of injuries has been detailed in the chapter on trauma. However, even in the absence of clear history of trauma, any bruises or contusions should be noted as they may have medicolegal implications in the future.

TABLE 1: Common dermatological lesions seen in critically ill patients.

Lesion	***Description***	***Image***	***Comments***
Macule	Flat, circumscribed, <1 cm diameter		Maculopapular rash in viral infections, cutaneous candidiasis
Patch	Flat, circumscribed, >1 cm diameter		Fixed drug rash, hypopigmented patches in leprosy
Papule	Circumscribed, solid, elevated, <1 cm diameter		Allergic reactions, viral rash—varicella
Plaque	Circumscribed, solid, elevated, >1 cm diameter		Pustular psoriasis, lupus vulgaris
Nodule	Circumscribed, solid, elevated, >1 cm diameter, depth>width		

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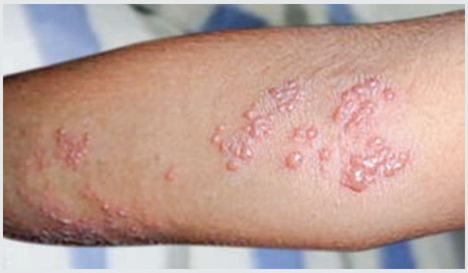
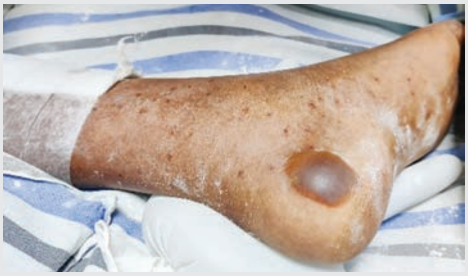
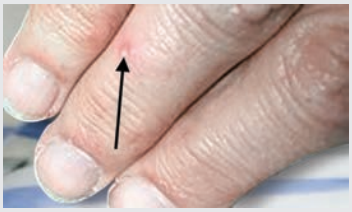
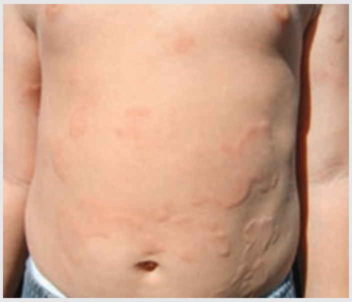
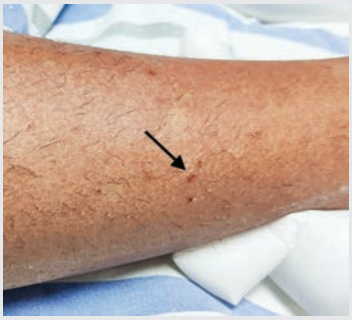
Lesion	***Description***	***Image***	***Comments***
Vesicle	Circumscribed, fluid-filled, elevated, <1 cm diameter		Herpes simplex, varicella, herpes zoster
Bulla	Circumscribed, fluid-filled, elevated, >1 cm diameter		Bullous impetigo, bullous drug reaction, friction blister, pemphigus vulgaris, late stages of necrotizing fasciitis
Pustule	Circumscribed, pus-filled, elevated, <1 cm diameter		Impetigo, pustular psoriasis
Wheal	Circumscribed, transient, edematous elevation of skin		Allergic reactions
Ulcer	Full thickness loss of epidermis, part of dermis, looks like a defect in the skin		Pressure sores, venous ulcer
Petechiae	Pinpoint round spots, can be red or purple		Dengue, TTP

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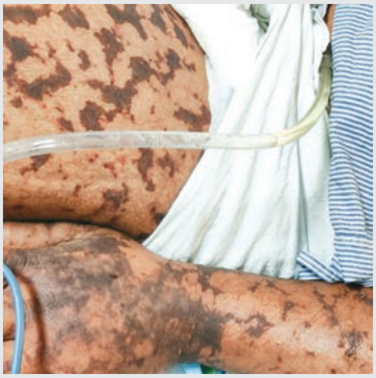
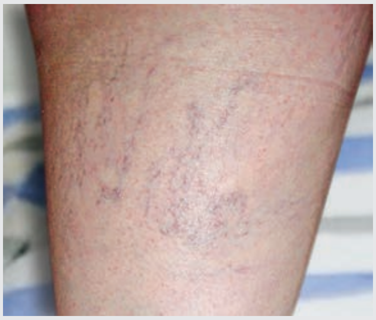
Lesion	*Description*	*Image*	*Comments*
Purpura	Purple-red lesions over skin; may or may not be palpable		Meningococcemia, rickettsial fever, TTP
Telangiectasia	Superficial dilation of capillary		Scleroderma, steroid abuse

(TTP: thrombotic thrombocytopenic purpura)

Presence of scars such as old surgical scars will give important information about previous procedures the patient may have undergone. Presence of scars of deliberate self-harm such as healed cuts over the wrist or concealed parts of the body must be noted as they may carry diagnostic value, as well as aid in diagnosis of psychological disorders. This can help secure necessary support for the patient in form of counseling or medication during their recovery. Excoriation marks over the skin may indicate presence of pruritis such as in a patient with obstructive jaundice.

There are times when skin changes result from primary skin diseases or secondary to underlying medical conditions. Primary changes have been listed in **Table 1**. These lesions may be accompanied by secondary changes such as:

- *Scaling:* Visible exfoliation, for example, exfoliative dermatitis
- *Crusting:* Dried serum or pus over lesion, for example, impetigo
- *Erosion:* Loss of superficial layer of skin, for example, herpes and varicella.

Palpation

Following this, the next step is palpatory examination. One must examine the lesions with a gloved hand to look for any blanching, tenderness, or pain over lesions, edema of skin, and to look for signs of desquamation.

Blanching is usually elicited by pressing the fingertip against the skin for 3–4 seconds. This is accompanied by a decrease in the redness or erythema, which reappears when the finger is removed. Diascopy is a slightly more refined way to demonstrate this sign. A piece of clear glass or plastic is used to apply pressure over the affected area. If the redness is because of dilated blood vessels (such as in an erythematous, blanching rash), the underlying skin will become pale and erythema will reappear as the pressure is reduced. A nonblanching rash shows no change in color.

Desquamation can be confirmed if lateral pressure over the lesion results in sloughing of skin. This is known as Nikolsky positive and may indicate a very serious rash such as SJS or immunobullous disease.

Palpation also draws attention to differences in temperature. For example, if the lesion is secondary to local inflammation, the area may be warm. Arterial insufficiency may be indicated by a decreased temperature of the affected area. All peripheral pulses should be palpated. Differences in pulses or absent pulses may indicate embolic phenomena or vascular insufficiency due to thrombosis or dissection.

Urticarial rashes are raised, palpable, erythematous rashes (refer to erythematous rashes for details) and are characterized by a phenomenon called dermatographism. This is characterized by the appearance of wheals or urticarial rashes over skin when it is firmly stroked.

Diagnosis of the disorder is made based on the site of lesion, morphology of lesion, associated symptoms, and severity of systemic symptoms. There are different types of skin lesions, which are encountered in our daily practice. They can be classified in numerous ways—etiologically (as listed above), anatomically, and morphologically **(Table 1)**. As the current focus of our chapter is on clinical examination, we will be discussing the morphological classification of these lesions.

Approach to Rashes and Dermatological Lesions in Intensive Care Unit

This section of the chapter is aimed at the discussion of assessment of a case like this. Santistevan et al. described an approach to rashes in patients presenting to the emergency department based on visual and tactile pattern of the rash. Rashes can be divided as in **Figure 2**. A similar approach can be used for initial examination of ICU patients.

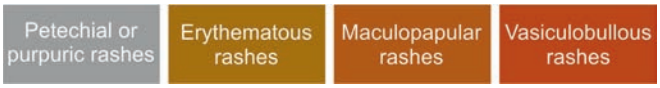


Fig. 2: Morphological classification of rashes.

Petechial or Purpuric Rashes

Petechiae are pin-point lesions, red-purple, and result from bleeding of capillaries into skin. They are nonblanching unlike erythematous blanching lesions seen in allergic reactions, telangiectasias, or exanthema. Larger lesions (>0.5 cm) are referred to as purpura. They may be palpable or nonpalpable. The diagnosis of the underlying condition is based on presence or absence of fever and on whether the rash is palpable or not. **Table 2** illustrates the possible diagnoses based on these criteria.

Purpura fulminans: It results from hemorrhagic skin necrosis and microvascular thrombosis. It is commonly seen in meningococcemia, streptococcal infection, staphylococcal, and rickettsial fevers. Viruses such as varicella have also been known to cause purpura fulminans.

It starts as an erythematous rash in dependent areas such as the ankles, as well as hands and wrists. Subsequently, there is formation of a maculopapular rash that becomes palpable and then the lesions coalesce to form purple-black colored purpuric lesions. Lesions are well-delineated and can involve the trunk also **(Fig. 3)**. It can result in extensive skin necrosis especially in extremities. The rash is nonblanching with formation of irregular areas of blue-black necrosis, which can extend to deeper skin layers **(Fig. 4)**. This is especially common in rash associated with rickettsial fever and concomitant high vasopressor requirement. In meningococcemia, skin cultures may reveal the organism.

Henoch–Schöenlein purpura: It is an autoimmune vasculitis commonly presenting with a palpable purpura over legs and buttocks. It is associated with gastrointestinal hemorrhage and acute-onset nephritis. It is more common

TABLE 2: Approach to diagnosis in petechial/purpuric rashes.

Palpable		***Nonpalpable***	
Febrile and toxic	Afebrile	Febrile and toxic	Afebrile
Meningococcemia	Autoimmune vasculitis	TTP	ITP
Disseminated gonococcal infection		DIC	
Bacterial endocarditis		Purpura fulminans	
Henoch–Schöenlein purpura		Fat embolism	

(DIC: disseminated intravascular coagulation; ITP: idiopathic thrombocytopenic purpura; TTP: thrombotic thrombocytopenic purpura)
Source: Adapted from Santistevan J, Long B, Koyfman A. Rash decisions: an approach to dangerous rashes based on morphology. J Emerg Med. 2017;52(4):457-71.

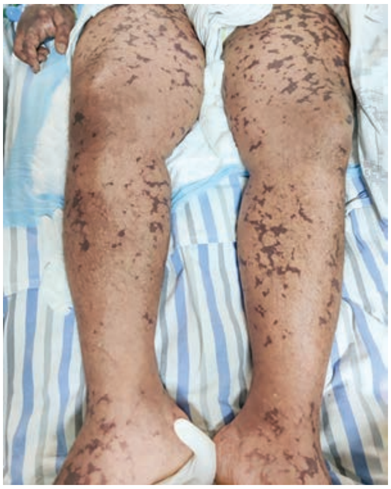


Fig. 3: Purpura fulminans.

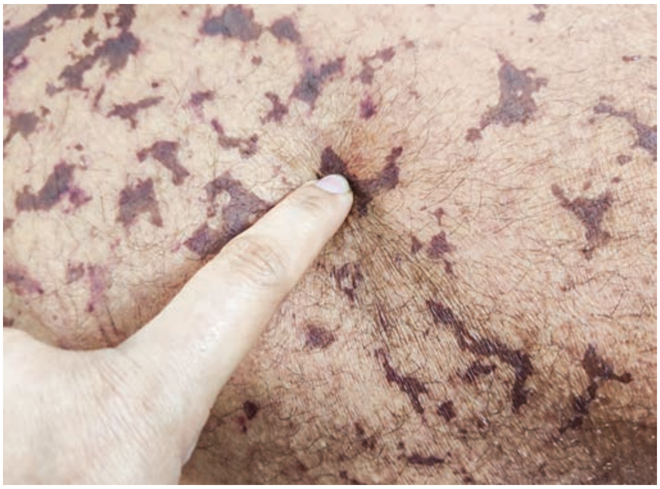


Fig. 4: Nonblanching rash of purpura fulminans.

TABLE 3: Classification of erythematous rashes.

Febrile and toxic		***Afebrilez***	
Nikolsky+	***Nikolsky–***	***Nikolsky+***	***Nikolsky–***
Staphylococcal scalded skin syndrome	Toxic shock syndrome	Toxic epidermal necrolysis	Anaphylaxis
Stevens–Johnson syndrome	Scarlet fever		Medications (niacin, vancomycin)
Toxic epidermal necrolysis			

Source: Adapted from Santistevan J, Long B, Koyfman A. Rash decisions: an approach to dangerous rashes based on morphology. J Emerg Med. 2017;52(4):457-71.

in children. Patients may also complain of abdominal pain and joint pain.

Bacterial endocarditis: Patient presents with fever, malaise, and rash. Common skin findings are presence of palpable purpura, splinter hemorrhages (lesions in nail bed), Osler nodes (nodules over pads of fingers and toes, which are painful and purple), and Janeway lesions (painless macules on palms and soles).

Fat embolism: Petechial rash is seen in 50% of patients with fat embolism. It is mostly visible in the face and upper torso. It is usually transient and disappears in 24 hours.

Thrombotic thrombocytopenic purpura (TTP) and disseminated intravascular coagulation (DIC) are both characterized by a nonpalpable petechial rash in a patient who may be febrile and toxic.

Erythematous Rashes

These rashes are typically diffuse, red skin changes, and result from capillary congestion. The presence of fever and toxic appearance may indicate the presence of a life-threatening rash. The diagnosis can further be arrived at by testing for the Nikolsky sign. **Table 3** will aid to narrow down the diagnosis.

Staphylococcal scalded skin syndrome: Usually presents in children with a red rash and sloughing of the epithelium. It is caused by strains of *S. aureus*, which release an exfoliative toxin. Rash begins in axilla, neck, groin, and is diffuse, erythematous, blanching type, and tender. There can be bulla formation in some instances. Cultures of bullae fluid will be sterile. Mucous membranes are not involved.

Stevens–Johnson syndrome and toxic epidermal necrolysis (TEN): They are also known as severe cutaneous adverse reactions. Both are the same, only extent of the lesions differ between the two. They are characterized by erythema followed by epidermal detachment resulting in bulla or blister formation. These breakdown to form erosions and result in skin necrosis. Lateral pressure causes skin detachment (Nikolsky positive). There is an involvement of mucous membranes including eyes, mouth, and genital area resulting in mucosal erosions. This can help differentiate it from staphylococcal scalded skin syndrome. A biopsy can also help reach a definitive diagnosis. Involvement of <10% is known as SJS, 10–30% is known as SJS/TEN overlap, and >30% is known as toxic epidermal necrolysis. The SCORTEN severity scale or ABCD-10 can be used for prognostication of patients with TEN.

DRESS syndrome: DRESS is a drug-induced reaction with multisystem involvement. It is characterized by an erythematous rash, which may also be maculopapular followed by desquamation. Commonly implicated drugs include antiepileptics (carbamazepine, phenytoin, lamotrigine, phenobarbital, and oxcarbazepine); antibiotics such as sulfonamides, vancomycin, minocycline, and nevirapine; and antitubercular drugs such as rifampicin and ethambutol and allopurinol. They are usually started 2–8 weeks before appearance of symptoms. It is associated with fever, lymphadenopathy, eosinophilia, and signs of organ involvement such as hepatitis, kidney involvement, pulmonary involvement (Dry cough, shortness of breath, wheeze, and infiltrates on chest X-ray), and cardiac involvement [hypotension, electrocardiogram (ECG) changes].

Toxic shock syndrome: If Nikolsky sign is negative and patient is toxic, one must consider the possibility of toxic shock syndrome arising from a staphylococcal infection. It is characterized by a diffuse erythematous rash, which subsequently desquamates over hands and feet. These patients develop severe vasodilatory shock, which is refractory to fluid resuscitation. Antibiotics and source control remain mainstay of treatment.

Anaphylaxis: Anaphylaxis can result from a drug reaction, food allergy, insect bites, or toxin exposure. It is associated with pruritis, diffuse erythema, swelling of mucous membranes such as lips, tongue, associated wheeze, stridor, and respiratory compromise or hemodynamic collapse **(Fig. 5)**. Presence of an urticarial rash is one of the features of anaphylaxis. Urticarial rash or wheals are elevated, palpable, pinkish lesions with pale centers and an erythematous flare. It is a blanching rash and can have various shapes—linear, serpiginous, or circular. Desquamation is not a feature and Nikolsky sign is negative. The lesions are usually transient and migratory in nature.

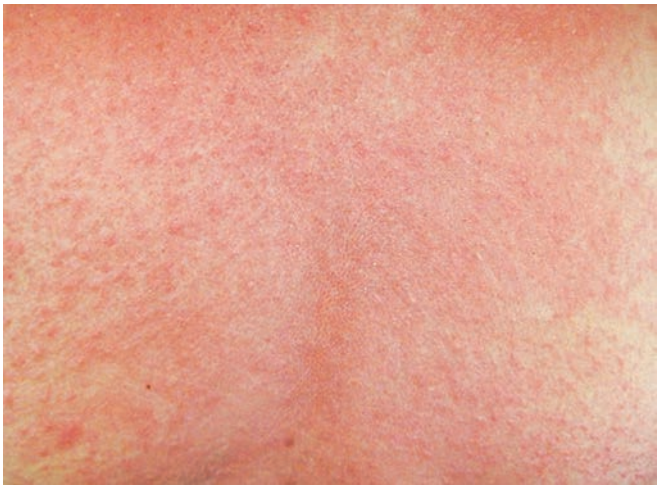


Fig. 5: Erythematous blanching rash over chest.
Source: Wikimedia Commons contributors. (2020). Rash on the chest of a person with anaphylaxis. Available from: https://commons.wikimedia.org/w/index.php? [Last accessed June, 2024].

Maculopapular Rash

This type of rash can be seen in a wide variety of situations including viral infections, tropical illnesses such as rickettsial fevers, autoimmune diseases, drug reactions, and disseminated bacterial infections.

Diagnosis starts from history, location of rash, distribution of rash, and severity of critical illness. Santistevan et al. classify the rash based on the severity of systemic illness and distribution, that is, central or peripheral **(Table 4)**.

Common viral exanthems:

- *Measles:* An initial prodromal phase of fever, malaise, cough, conjunctivitis, and anorexia is followed by development of a maculopapular rash after 2–4 days. Rash is blanching and starts over face, trunk, and then involves the peripheries. Patients can also develop buccal mucosal lesions opposite molar teeth called Koplik spots. These are white, blue or gray lesions, elevated, and with an erythematous base.
- Acute Epstein–Barr virus (EBV) infection can present with a maculopapular rash beginning over trunk and arms followed by spread to forearms and face.
- Dengue is associated with a faint macular, morbilliform, or scarlatiniform rash that spares the palms and soles.
- Human immunodeficiency virus (HIV) seroconversion is sometimes associated with a maculopapular rash

TABLE 4: Approach to maculopapular rash.

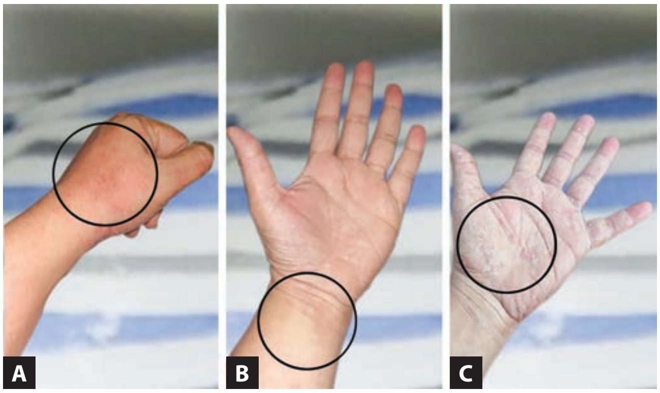
Central		***Peripheral***	
Febrile/toxic	Afebrile	Febrile/toxic	Afebrile
Viral exanthema	Drug reaction	Stevens–Johnson syndrome	Scabies
Measles	Pityriasis	Erythema multiforme	Eczema
Lyme disease		Rickettsial fevers	
		Lyme disease	
		Meningococcemia	

Source: Adapted from Santistevan J, Long B, Koyfman A. Rash decisions: an approach to dangerous rashes based on morphology. J Emerg Med. 2017;52(4):457-71.

affecting palms and soles of the feet. There may also be papules/nodule formation over trunk.

- Although quite rare, influenza viral infections are known to be associated with a maculopapular rash, which also spares palms, plantar surfaces, and face.

Erythema multiforme: Erythema multiforme is an autoimmune reaction in response to drugs (sulfa drugs, antibiotics, and anticonvulsants), viral infections such as herpes simplex, fungal infections, or mycoplasma. It is characterized by pruritic maculopapular lesions, which may be targetoid. They usually involve extensor surface of extremities, palms, soles, dorsa of hands, feet, and face **(Figs. 6A to C)**. They last for 1–2 weeks. Erythema multiforme minor is limited to skin rash, however, erythema multiforme major is associated with mucosa involvement and systemic symptoms, which may be life-threatening.



Figs. 6A to C: Erythema multiforme following chloroquine compound consumption. (A and B) Erythematous maculopapular rash over dorsum and palmar aspects of hand; (C) Desquamating rash over palm after 1 week.

Secondary syphilis: It presents as a systemic illness associated with a maculopapular rash over trunk and extremities, including palms and soles. Lesions are copper colored or reddish-brown.

If the patient remains afebrile, one must think of other causes such as drug reactions, scabies, or psoriasis.

Vesiculobullous Rashes

Vesicles and bullae occur due to damage at the level of the dermoepidermal junction. Approach to diagnosis can be classified based on rash distribution and presence or absence of fever as elaborated in **Table 5**.

TABLE 5: Approach to vesiculobullous rashes.

Diffuse		***Localized***	
Afebrile	Febrile	Afebrile	Febrile
Bullous pemphigoid	Varicella	Herpes zoster	Necrotizing fasciitis
Pemphigus vulgaris	DIC	Contact dermatitis	

(DIC: disseminated intravenous coagulation)
Source: Adapted from Santistevan J, Long B, Koyfman A. Rash decisions: an approach to dangerous rashes based on morphology. J Emerg Med. 2017;52(4):457-71.

Necrotizing fasciitis: It can begin as erythema and swelling of the affected area with extreme pain. Initially pain may be greater than the severity seen on inspection of the area. Commonly affected areas include extremities, genitalia, and perineum. As the necrosis worsens, patient becomes more toxic and develops vesicles or bullae, which can rupture. More details have been discussed in the segment below on skin and soft tissue infections.

Varicella: It is characterized by a rash, which appears in crops and evolves from papules to vesicles, which rupture and develop crusting. Lesions have an erythematous base and present in a number of stages at one time, unlike smallpox where the lesions are in the same stage at a particular time.

Herpes zoster vesicles: They have dermatomal distribution and are painful. The pain is severe and usually precedes the appearance of the rash. It begins as erythematous papules and progresses to form vesicles, which are grouped and have an erythematous base. Patients can have associated organ involvement such as hepatitis, meningoencephalitis, and pneumonia. Patients can also develop herpes zoster ophthalmicus, keratitis, uveitis, herpes zoster oticus, and motor neuropathy.

Bullous pemphigoid: It is an autoimmune skin disease characterized by blister formation. It usually affects the elderly (>65 years). A prodromal phase lasting weeks to months, characterized by development of papules, eczema or urticarial skin lesions precedes the development of the blisters. Subsequently, tense blisters and bullae develop. Flexural areas are most commonly involved including extremity folds, axillary and inguinal folds, and trunk. Mucosa is involved in 10–30% patients. The rash is diffuse and these patients are usually afebrile and nontoxic.

Pemphigus vulgaris: It is also an autoimmune disease with formation of blisters, which are superficial and flaccid. It affects large areas with painful erosions and sloughing of skin. Physiologically, they behave like burns patients and suffer fluid loss. Nikolsky sign is positive.

Back to the Case Scenario

Our patient presented with a history of fever and cough. The appearance of an erythematous rash and giddiness was following the consumption of amoxicillin clavulanate. Differentials for such a rash would be viral exanthems, rickettsial diseases (though rash is more peripheral than central), and an allergic reaction. This patient may be having an allergic reaction to the antibiotic. The presence of pruritis over the rash, tachycardia, hypotension, and bronchospasm further suggest that the patient is suffering from anaphylactic shock secondary to amoxicillin clavulanate.

Other Skin Lesions Encountered in Critically Ill Patients

Eschar

Being a tropical country, we see a lot of patients with rickettsial diseases. Eschars are necrotic patches, which occur at the site of mite or tick bite. An eschar usually begins as a papule, which is small and painless and forms a few days after the bite. It subsequently becomes pustule or vesicular, develops a red, annular halo, and ulcerates forming a punched-out ulcer with black-brown crusts. It resembles a cigarette burn **(Fig. 7)**.

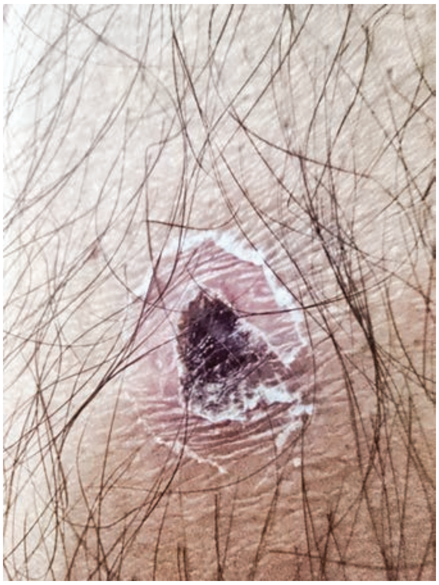


Fig. 7: Eschar seen in rickettsial infections.

The presence of an eschar is pathognomonic for rickettsial fever, and it is seen in 7–97% of cases. It is usually present in moist and intertriginous areas such as axilla, groin, perineum, neck (can be hidden by the hairline), chest, and abdomen. It is usually single. It is accompanied by a maculopapular rash in some instances. In severe cases, patients develop purpura fulminans as discussed earlier.

Ecthyma Gangrenosum

It is a skin infection characterized by ulcers with central necrotic areas appearing over extremities, axillary, and anogenital areas. It results from perivascular invasion and necrosis of the overlying skin. It occurs due to fulminant bacteremia especially in immunocompromised patients and has been described most commonly in *Pseudomonas aeruginosa* infections. It starts as painless, erythematous papules, and macules and evolves to a hemorrhagic vesicle and subsequently evolves into a gangrenous ulcer with an eschar. Treatment involves wound care, antibiotics, and source control. Differential diagnoses are listed in **Box 1**.

Hyperpigmentation of the Skin

Brown-black pigmentation depends on the activity of melanocytes. Brown pigmentation can also be due to hemosiderin deposition (hemochromatosis). Generalized hyperpigmentation, more in flexural areas, is seen in Addison disease.

Examination of the skin and nails is also important. Thinning of hair can be seen in autoimmune diseases. Nails can show splinter hemorrhages such as in bacterial endocarditis.

BOX 1: Differential diagnosis of ecthyma gangrenosum.

- Autoimmune vasculitis
- Vasopressor-induced necrosis
- Calciphylaxis
- Disseminated intravascular coagulation
- Pyoderma gangrenosum
- Necrolytic migratory erythema

TABLE 6: Grades of severity of pressure sores.

Stage	*Description*
Stage 1	Intact skin with nonblanchable erythema
Stage 2	Loss of partial thickness skin, intact dermis
Stage 3	Loss of full thickness skin
Stage 4	Full

Pressure Sores or Pressure Injuries in Intensive Care Unit

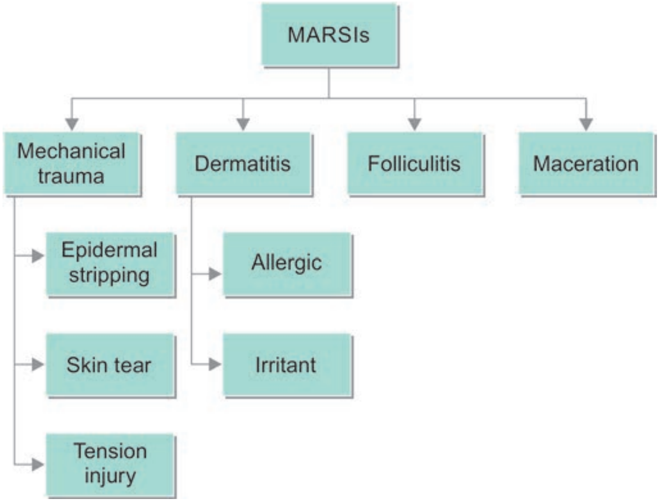
During initial assessment, presence of any pressure wounds must be documented. Assessment of skin for pressure-related injuries should take place every shift. Sometimes, this may need to be more frequent, especially if the patient is at a greater risk for pressure sores. Pressure ulcers usually occur over bony prominences such as sacrum, coccyx, trochanteric region, heels, elbows, and ears. In patients who have been proned for long durations and where position change is a challenge, pressure sores can occur over the face, breasts, and abdomen. The grades of pressure sores have been described in **Table 6**.

Pressure injuries can also result from medical equipment such as ECG wires or tubings crossing the body.

Healthcare-associated injuries include medical adhesive-related skin injuries, which can range from erythema to formation of erosions, vesicles, blisters, or skin rupture persisting for >30 minutes after adhesive removal. They can be classified as shown in **Flowchart 1**. Presence of such skin changes must be regularly documented and necessary treatment must be given.

The best approach toward pressure injuries is their prevention. Everyday examination should include paying heed to important measures being taken in prevention of pressure injuries. These include functional airbeds and water beds, air-filled gloves placed strategically at areas prone for pressure sores; proper equipment while proning such as silicone gel-based head rests, head rings, and chest rolls; and proper padding given over susceptible areas. It is also important to avoid prolonged contact with moisture and ensure adequate frequency of position change.

Flowchart 1: Classification of medical adhesive-related skin injuries (MARSIs).



PART II: EXAMINATION OF SKIN AND SOFT TISSUE INFECTIONS (SOFT TISSUE)

CASE SCENARIOS

1. *A 60-year-old male, farmer by occupation, came with complaints of pain in the left foot with redness and swelling since 3 days. He is a poorly controlled diabetic on oral hypoglycemic agents (OHAs). On arrival at the Emergency, he was found to be drowsy, tachycardic, tachypneic, and hypotensive. His arterial blood gas (ABG) showed severe metabolic acidosis. He was intubated and shifted to the ICU for further care.*
2. *A 55-year-old female, known case of stage 4 carcinoma breast, presented with history of pain and swelling in the right lower limb since 3 days and breathlessness for 1 day. On arrival to the emergency, she was found to be drowsy, tachycardic, tachypneic, and hypotensive. Her ABG showed severe metabolic acidosis. She was intubated and shifted to the ICU for further care.*

Cases 1 and 2 are similar with a few minor differences. Both patients are likely suffering from an infection of the skin/soft tissue. A good and detailed history is imperative to making an accurate diagnosis. They need to undergo a detailed examination—both local and systemic—so that important findings are not missed.

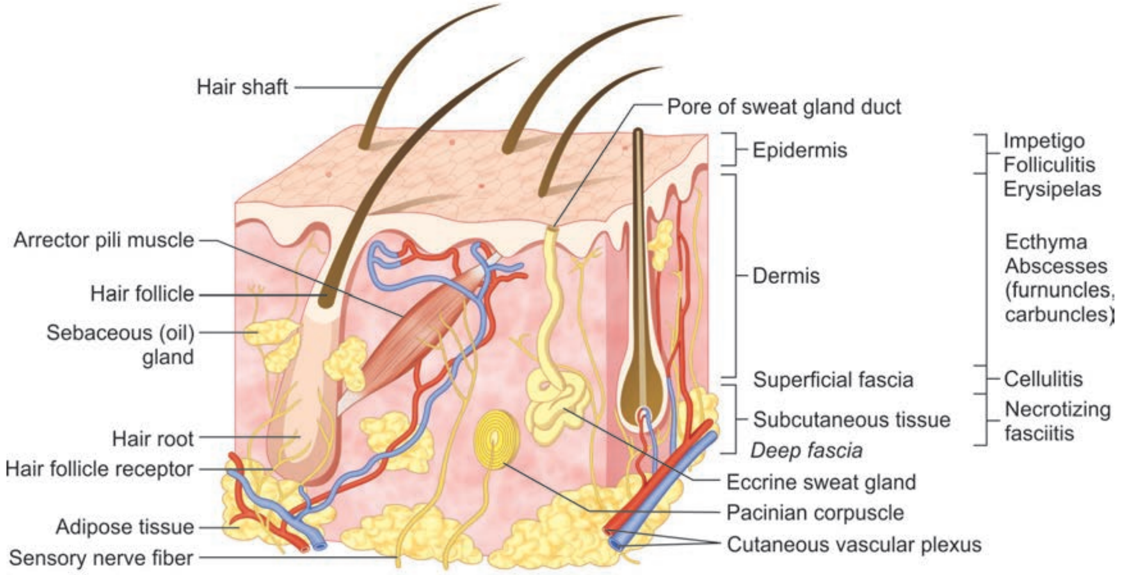

Hair shaft
Pore of sweat gland duct
Epidermis
Impetigo
Folliculitis
Erysipelas
Arrector pili muscle
Hair follicle
Sebaceous (oil) gland
Dermis
Ecthyma
Abscesses (furnuncles, carbuncles)
Superficial fascia
Cellulitis
Subcutaneous tissue
Necrotizing fasciitis
Deep fascia
Hair root
Hair follicle receptor
Eccrine sweat gland
Pacinian corpuscle
Adipose tissue
Sensory nerve fiber
Cutaneous vascular plexus

Fig. 8: Soft tissue infections classified based on layers of skin.
Source: Modified from Wikimedia Commons. (2023). Structure of the skin. [online] Available from https://commons.wikimedia.org/w/index.php?title=File:501_Structure_of_the_skin.jpg&oldid=789279398 [Last accessed June, 2024].

Skin and soft tissue infections have been classified in various ways depending on the etiological agent, rate of progression, severity, clinical presentation, and skin extension. One method is to classify based on the number of skin layers involved **(Fig. 8)**.

History-taking and Symptomatology

Duration of symptoms is an indicator of type of pathology. Swelling of the limbs or a particular area is a common symptom. Acute swelling of <72 hours duration is more commonly associated with deep vein thrombosis, infection, trauma, allergic reaction, envenomation, or heart failure. Chronic swellings are usually >3 months duration and may be due to long-standing medical diseases such as chronic kidney disease, chronic liver disease, or other causes such as venous insufficiency or lymphedema. Exacerbation of endocrine diseases such as hypothyroidism or cardiorespiratory conditions such as cor pulmonale can also precipitate swelling of limbs.

The depth of the infection and presence or absence of necrosis are usually used to classify and prognosticate skin and soft tissue infections **(Fig. 8)**.

Patients with cellulitis usually present with pain, redness, and swelling of the affected site, which is acute in onset. Pain is a very important symptom and detailed history regarding site, onset, duration, aggravating, and relieving factors is necessary. Extremely severe pain preceding appearance of skin lesions may be seen in acute vascular insufficiency and NF. Claudication pain is a muscle pain usually aggravated by walking or standing and relieved by rest. It is commonly due to peripheral vascular disease. Intermittent pain can also be neurogenic due to nerve compression along the spinal cord. This is usually relieved on sitting and is known as "pseudoclaudication". Pain may or may not be preceded by history of trauma—trivial or significant. Pain is a very important symptom as it not only points to the diagnosis but also the severity. Pain, which is disproportionate to the findings of clinical examination, is a typical feature of NF. In NF, pain also extends beyond the margins of the skin involved.

Other differentials for unilateral, painful, lower limb swelling should be considered while taking history such as deep vein thrombosis, lymphatic obstruction causing lymphedema (usually painless), trauma/fractures if patient is unconscious, thrombophlebitis, septic arthritis, or acute arterial occlusion. Risk factors for deep vein thrombosis include recent major surgery, trauma, prolonged immobilization following surgery or a long journey, history of malignancy, or chronic venous insufficiency. Presence of associated fever may indicate either a local infection or a noninfectious cause such as a deep vein thrombosis or malignancy. Abdominal masses can produce venous compression resulting in unilateral or bilateral lower limb swelling. History of joint involvement and restricted limb movement should be elicited while keeping in mind the possibility of septic arthritis, ruptured bursa, an intra-articular hematoma, or intramuscular hematoma.

Relevant history must be taken to evaluate for specific etiologies including history of trauma, history of an insect bite/snake bite, animal bites, or walking barefoot. History of any chronic vascular insufficiency should also be taken; one must ask about the presence of any claudication pain (see above) or history of varicose veins or any recent limb surgeries. History of having consumed undercooked seafood or raw fish must be taken from patient, as this can lead to NF caused by *Vibrio* spp, *Aeromonas*, and *Shewanella*. Any trauma involving salt water can predispose to infection by *Vibrio vulnificus*, whereas exposure to fresh or brackish water, contaminated soil, or leech use may result in NF by *Aeromonas hydrophila*. Consumption of raw or undercooked seafood can also result in NF. History of recent surgeries such as that for a perforated appendix, perforated diverticulitis, colon cancer with an obstruction, or complicated intra-abdominal infection may predispose to NF.

Past history will include history of any operative procedures and chronic disease such as diabetes, or heart failure, or cancer. Diabetes predisposes patients to methicillin-resistant *Staphylococcus aureus* (MRSA) and NF. Diabetes also results in neuropathy predisposing patients to trivial trauma, which may go unnoticed, associated with impairing wound healing. History of past tuberculosis should also be taken. Immunocompromised state may weaken host defenses and result in greater susceptibility to bacterial invasion. The presence of recent hospitalization or antibiotic use may indicate infection by resistant bacteria.

Going Back to Our Case Scenario

Both patients presented with unilateral and painful limb swelling. Attention to the past history can give valuable clues. While the patient in Case 1 has uncontrolled diabetes, the patient in Case 2 is suffering from metastatic carcinoma breast. While cellulitis may seem to be the most obvious diagnosis, unilateral deep vein thrombosis must be considered in the differential, especially in Case 2. It can be ruled out by venous compression ultrasound.

Examination

Vitals of the patient are recorded prior to proceeding with systemic or local examination. Presence of fever, tachycardia, and hypotension suggest a deeper skin or soft tissue infection, which is usually more severe. However, even superficial lesions such as those seen in staphylococcal toxic shock syndrome may be associated with severe systemic manifestations. Once the patient's vitals are noted and a head-to-toe examination is done, we proceed to a local examination of the affected area.

Inspection

On approaching the patient, one must introduce themselves. Bedside nurse should be involved in patient examination. Hand hygiene should be performed prior to examination. Curtains must be closed to ensure adequate privacy. After informing the patient and taking verbal consent, the affected area should be completely exposed. This will help delineate the exact extent of the lesion/s.

The classical findings of inflammation: Dolor (pain), calor (heat), rubor (redness or erythema), and tumor (swelling) are used to diagnose the presence of skin and soft tissue infections. Fluor or production of secretion has been added as a fifth component. Functio laesa or loss of function is usually a result of pain and swelling. Redness and swelling are usually apparent on inspection. Presence of secretions or discharge from the wound should be noted and recorded. Loss of function such as inability to move the limb may also be observed during inspection. This may indicate either severe pain, vascular insufficiency, or nerve damage. Presence of an IV cannula at the site of skin changes may indicate the possible etiology of surrounding limb swelling, that is, thrombophlebitis or even drug extravasation. Patients with cellulitis have unilateral swelling with erythema. Presence of skip lesions, blisters, necrotic patches could indicate presence of NF (see below).

The color of the surrounding skin may also give an idea of the vascularity. In acute vascular insufficiency, the skin may initially look pale and subsequently cyanosed and gangrenous changes may develop **(Fig. 9)**.

Features such as dry skin, loss of hair, nonhealing ulcers, nail pigmentation, and thickening and blisters over foot are indicative of a peripheral neuropathy. Chronic changes such as thick, wrinkled, and hyperpigmented skin are seen in patients with long-standing inflammation, edema, or chronic infection.

Some abscesses may rupture and present as a discharging sinus. When such a sinus is close to a joint or bone, it should be checked for underlying osteomyelitis/extension into the joint using clinical examination and imaging.

Palpation

Further examination is undertaken with gloved hands **(Fig. 10A)**. Careful palpation may reveal information about tenderness, which indicates underlying inflammation. Severe tenderness extending beyond the margins of inflammation is seen in NF. Absence of sensations or reduced sensations in a swollen limb, especially in the background of trauma may indicate damage or compression of a nerve. It may also indicate underlying peripheral nerve damage such as in diabetes.

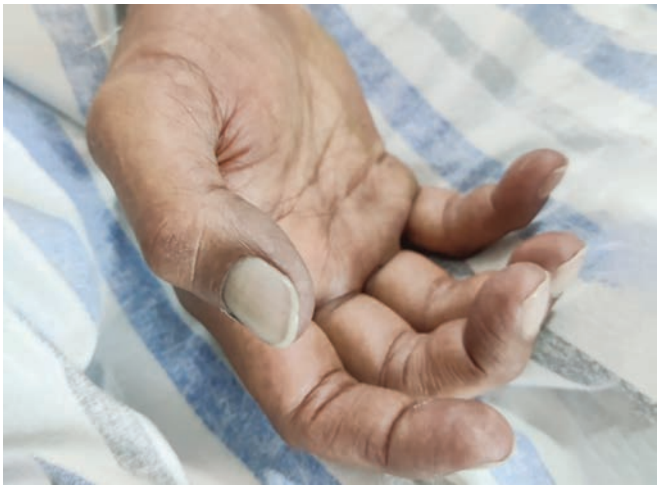


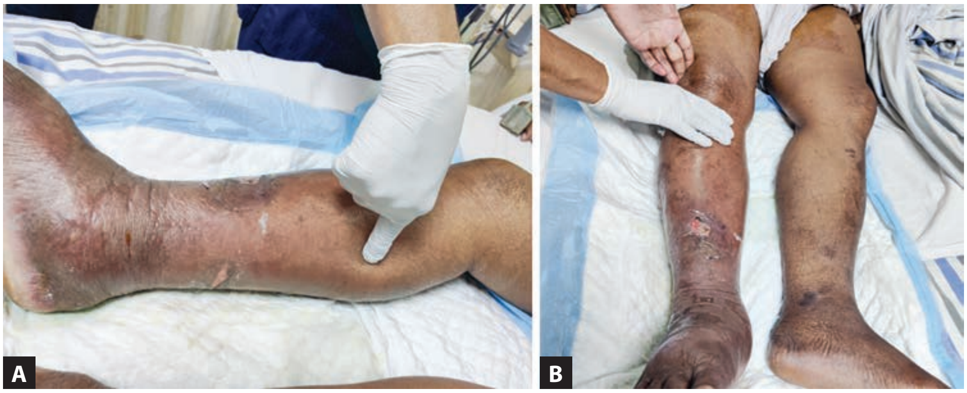
Fig. 9: Peripheral cyanosis in acute vascular insufficiency.

If there is associated erythema, palpation may reveal presence of blanching or nonblanching erythema **(Fig. 10A)**. Cellulitis presents with blanching erythema, swelling, pain, and localized warmth. The adjacent joint should also be examined **(Fig. 10B)**.

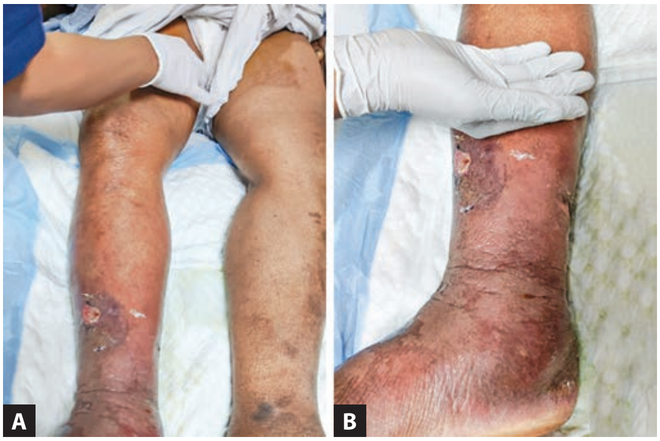
Palpation may also reveal the presence or absence of edema or swelling and the actual extent of the lesion **(Fig. 11A)**. Eliciting calf tenderness by palpation may indicate the presence of a deep venous thrombosis, which forms a differential diagnosis for soft tissue infection of the limb.

Palpation gives information about temperature differences between the limbs **(Fig. 11B)**. An extremely warm limb indicates ongoing inflammation such as in cellulitis. An extremely cold limb may indicate acute vascular insufficiency. All peripheral pulses must be palpated if this is suspected. In cellulitis, the area is usually inflamed, erythematous, and warm to touch. Palpation may reveal an area of hardness in the skin known as induration. Deep-seated abscess may present in this manner with pain and overlying induration **(Fig. 11A)**. This may indicate an underlying infective/inflammatory process such as an abscess or even malignant infiltration of the skin.

Sometimes skin palpation may reveal crepitus, which indicates the presence of air in the subcutaneous plane or subcutaneous emphysema. It feels like palpating "crumpled wrapping paper." In the presence of redness and inflammation, it could indicate the presence of gas forming organisms such as *Clostridia* sp. In trauma, crepitus can result from underlying pneumothorax, pneumomediastinum, tracheal injury, or hollow viscous perforation. If close to a joint, it should be examined for swelling, tenderness, or extension of the infection into the joint **(Fig. 10B)**.



Figs. 10A and B: (A) Presence of blanching erythema on palpation; (B) Examination of adjacent joint for swelling, tenderness, warmth, crepitus, and extension into joint.



Figs. 11A and B: (A) Palpation using gloved hand for extent of lesion and tenderness; (B) Examination of temperature of affected and surrounding areas using back of the gloved hand.

Adjuncts to Examination

The bedside ultrasound is a useful adjunct and a quick screening can give valuable information. It can give preliminary information regarding presence of deep venous thrombosis, pulsatile flow through arteries, and even visualize collections in the subcutaneous planes with some amount of training before a definitive imaging report can be given by the radiologist. This can be valuable in guiding management and patient stabilization while other resources and man power are mobilized.

Common Skin Infections Encountered in Critically Ill

Impetigo and erysipelas are superficial forms of infection. Impetigo can be bullous or nonbullous. Bullous impetigo is characterized by a fragile, flaccid, pus-filled bulla, which ruptures leaving behind a honey-colored crusted lesion **(Fig. 12)**. Most common areas affected include axilla, trunk, and intertriginous areas. One must also look for any pus-filled lesions or deep-seated abscesses, which may need emergent drainage.

Cellulitis is an acute, deeper infection, which spreads rapidly. It affects the skin and subcutaneous tissue and may need surgical debridement. While the lesions in erysipelas are bright red, superficial, and clearly demarcated, cellulitis is deeper and poorly demarcated. A close inspection of the area should be done to look for possible sources of infection such as breaks in the skin, nonhealing wounds or ulcers, and animal or insect bite marks. Findings that suggest a more serious infection and possible need for surgical intervention include presence of bullae, crepitus (may indicate gas gangrene), purpura, fluctuance, or presence of necrosis.

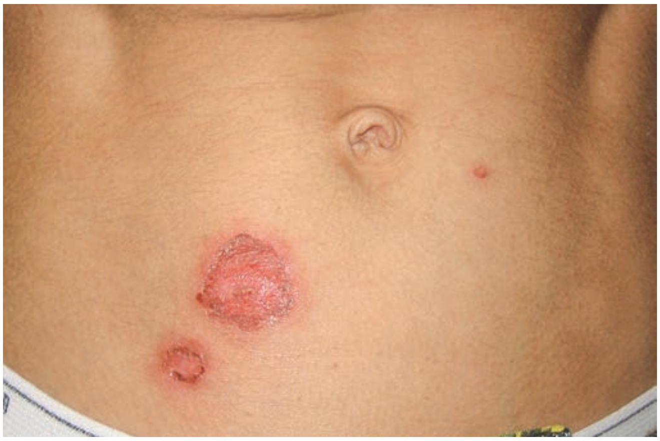


Fig. 12: Bullous impetigo.
Source: Wikimedia Commons. (2024). Bullous impetigo. Available from: https://commons.wikimedia.org/w/index.php?title=File:Bullous_impetigo1.jpg&oldid=838055780 [Last accessed June, 2024].

Necrotizing Fasciitis

Necrotizing fasciitis is a deep infection, which may extend to the muscle layer and endanger both limbs and life. It needs emergent intervention. It begins in the superficial fascia or hypodermis. Superficial layers are not affected in the early stages. It is more common in men and mortality is almost 100% if left untreated. Trauma is a common etiology.

Early symptoms include swelling, erythema, local warmth, and edema. Pain is one of the characteristic features of NF. Pain is usually disproportionate to the examination findings and manifests before the cutaneous findings. Presence of "skip lesions," that is, affected areas interspersed by normal looking skin and necrotic patches point to a diagnosis of NF. The affected area is indurated and tender but the margins of erythema are irregular and expanding. In NF, erythema extends beyond the margins of the swelling. Skin fluctuance is another characteristic feature. If the patients presents with advanced disease, they may demonstrate bullae and blisters on examination **(Fig. 13)**. The bullae may be filled with serous or hemorrhagic fluid. The fluid inside is characteristically describe as "dishwater discharge." These can further progress to rupture and form necrotic patches. Presence of crepitus on palpation indicates the presence of a gas-forming organism such as *Clostridium perfringens*.

In case of diagnostic uncertainty, the finger probe test has been described. NF is a clinical diagnosis and in case of diagnostic uncertainty, examination of the subcutaneous structures is carried out under local anesthesia. An incision is made in the anesthetized region extending up to the deep fascia in a sterile manner and a sterile, gloved index finger is used to probe the area. The test is considered positive for necrotizing fasciitis if the following three are present: (1) Absence of bleeding, (2) minimal pressure results in friable tissue, and (3) gray-colored "dishwater" discharge from the wound.

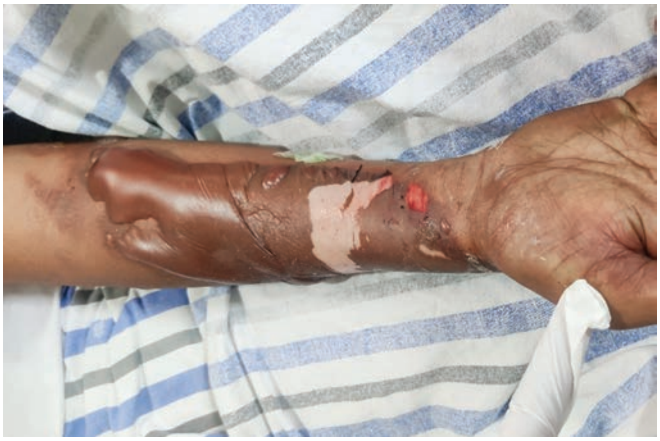


Fig. 13: Advanced necrotizing fasciitis with necrotic patches and bullae.

There are four types of NF classified based on the organism involved **(Table 7)**.

When the infection extends to the muscle planes, it is known as pyomyositis. When the infection involves the perineum, it is known as Fournier gangrene. These conditions require emergent antibiotics and extensive surgical intervention and carry a high mortality.

Surgical Site Infections

Surgical wounds, drain insertion, and invasive line site infections must be examined every day for signs of infection or inflammation. These include increase in swelling, erythema, purulent discharge, or tenderness. Any change is appearance of the wound must be addressed by involving the surgical team emergently.

Table 8 summarizes history taking in patients with skin and soft tissue infections. **Table 9** summarizes examination and common findings in various organ systems of these patients.

TABLE 7: Types of necrotizing fasciitis.

Type	***Organism involved***	***Common site***	***Predisposing factors***
Type 1	Polymicrobial (aerobic and anaerobic)	Trunk and perineum	Diabetes mellitus
Type 2	Monomicrobial and caused by beta hemolytic streptococci or MRSA	Limbs	
Type 3	Monomicrobial by *Clostridium perfringens* (Gas gangrene)	Limbs, trunk, and perineum	Trauma
Type 4	*Candida* spp. and zygomycetes	Limbs, trunk, and perineum	Immunosuppression

(MRSA: methicillin-resistant *Staphylococcus aureus*)

TABLE 8: Summary of history and symptomatology in skin and soft tissue infections.

Domain	***Relevant history to note***	***Comments***
Pain and swelling	• *Pain:* – Pain beyond visible lesion – Calf pain – Claudication—intermittent pain (see text) • *Swelling:* – Site – Extent and progression	• Arterial versus venous versus infective can be differentiated based on history. Pain due to complete acute arterial occlusion or an infection is severe and continuous • Pain due to venous congestion is dull aching and increases on walking/standing for long. Chronic venous insufficiencies are prone to venous ulcers and secondary infections

Contd…

Contd…

Domain	***Relevant history to note***	***Comments***
Temporal events	• *Current/recent illness:* – Renal failure, heart failure (pedal edema), infective endocarditis – Trauma, pricks from needles, insect or snake bites – Recent hospitalization and prolonged immobilization – Recent travel • *Recent surgery or instrumentation:* Major surgery or minimally invasive surgery	• The temporal events are important and can have etiological relevance and guide treatment • Necrotizing fasciitis can be associated with recent surgery, which is a known risk factor
Gastrointestinal (GI)	• Loose motions • Constipation and abdominal distension • GI malignancies	• Sometimes patients with cellulitis may present with GI symptoms • Infections with *Enterococcus*, *Streptococcus*, and *E. coli* can have GI symptoms • Constipation may indicate associated intestinal obstruction • In cases of extraperitoneal perforation—patient may present with thigh abscess
Fever	• *Infectious causes:* Cellulitis, necrotizing fasciitis, abscess • *Noninfectious causes:* Deep vein thrombosis, underlying malignancy, allergic reaction	
Altered sensorium	Severe infections can result in septic encephalopathy	Hyponatremia is common and a marker of severity and it may cause altered sensorium
Sepsis-induced organ failure	Can present in various ways including low urine output, jaundice, and new-onset dyspnea, based on the organ involved	

TABLE 9: Summary of examination of patients with skin and soft tissue infections.

Domain	***Eliciting of signs/symptoms***	***Comments***
Head, face, and neck	• Periorbital edema in renal failure • Anemia—hemolysis, blood loss (hemorrhage and hypotension), reduced production (CKD) • Jaundice—hemolysis, sepsis-induced cholestasis • Rash—malar rash in SLE, viral exanthems such as measles • Mask-like face in scleroderma with dermal induration and typical sharp features • *Mouth:* – Dry mucus membranes (hypovolemia) – Oral ulcers (sepsis and CTDs) – Lesions in erythema multiforme major – Narrowed aperture—microstomy in scleroderma • *Fundus:* Hypertensive retinopathy, diabetic retinopathy, signs of systemic infections such as TB/CMV • *Dilated neck veins:* Elevated JVP in congestive heart failure • Spider nevi over upper chest and neck—sign of chronic liver disease	This gives a quick clue regarding systemic diseases, acute vs. chronic illness and causes of oliguria/renal failure
Extremities	• *Fingers and palms:* – *Nail and digits:* Splinter hemorrhages, digital infarcts—IE, vasculitis – *Koilonychia:* Iron deficiency anemia – Osler nodes, Janeway lesions—infective endocarditis • *Hand and legs:* – *Reduced skin turgor:* Poor hydration status – *Rash:* Thrombocytopenia, Henoch–Schoenlein purpura, sepsis, TTP/HUS – *Pedal edema:* Pitting edema – *Cellulitis, necrotizing fasciitis:* Inflammation with necrotic patches, bullae, tender limb swelling – *Thickened, hyperpigmented skin over lower limbs:* Chronic skin changes due to venous insufficiency or venous congestion	Peripheral signs of vasculitis and infective endocarditis are easy to pick-up

Contd…

Contd…

Domain	*Eliciting of signs/symptoms*	*Comments*
Back and Perineum	• *Rash:* Rash over buttocks—Henoch–Schoenlein purpura • *Genital ulcers:* Systemic diseases and infection • *Sacral edema:* More relevant in supine patients for dependent edema • Excoriations in perineum and pressure sores due to chronic loose stools • *Perineum:* Common site for candida infection • Fournier gangrene is a life-threatening condition affecting the perineal region • Inguinal lymph node enlargement due to local infections or systemic diseases such as TB/HIV	The perineal and back examination is often missed in ICU patients that may give a clue of source of sepsis
Bedspace and documents	• Vasopressors, NTG infusions • Blood sugars • Urine bag—color quantity, trend of urine per hour	A careful bedspace examination is helpful in getting the trends
Cardio-vascular	• JVP, S3, S4, apex beat position—signs of chronic heart failure • *Signs of tissue hypoperfusion:* Cold peripheries, discoloration, increased CRT, peripheral cyanosis • High vasopressor support—digital gangrene	
Central nervous system	• Altered sensorium • *Muscle weakness:* Proximal weakness may indicate presence myositis as a part of autoimmune disease • *Peripheral neuropathies:* Diabetic neuropathy such as seen in trophic ulcers	Altered mental status may be due to septic/metabolic encephalopathy in severe infections
Respiratory	• Extrapulmonary ARDS may result in hypoxic respiratory failure presenting as hypoxia, bilateral crepitations • Systemic manifestations of CTDs may include DAH/interstitial lung disease	
GI	• Look for dilated veins or sinuses • Hernial orifices	

(ARDS: acute respiratory distress syndrome; CKD: chronic kidney disease; CMV: cytomegalovirus; CRT: capillary refill time; CTD: connective tissue disease; DAH: diffuse alveolar hemorrhage; GI: gastrointestinal; HIV: human immunodeficiency virus; HUS: hemolytic uremic syndrome; ICU: intensive care unit; JVP: jugular venous pressure; NTG: nitroglycerin; SLE: systemic lupus erythematosus; TB: tuberculosis; TTP: thrombotic thrombocytopenic purpura)

SUMMARY

Skin examination is a crucial part of patient assessment and should be done routinely for all critically ill patients. Skin manifestations can appear rapidly and sometimes be fleeting. Proper documentation of these in the case file will provide diagnostic clues and aid in better patient management.

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Chapter

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Postrecovery at Follow-up Clinic: What and how to Examine?

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> *"It is more important to know what sort of person has a disease than to know what sort of disease a person has."*
>
> —***Hippocrates***

ABSTRACT

In this chapter, we will focus on the approach for evaluation at follow-up after critical illness. To keep things practical, a case scenario is discussed with most possible clinical presentation. We have covered concepts behind principles of history taking at the follow-up, highlighting some unique differences of history taking from acute presentation. We further discuss relevant clinical examinations.

INTRODUCTION

There are many medical, psychological, and social challenges encountered by the survivors and caretakers of patients admitted to the intensive care unit (ICU) after hospital discharge. The goal of examining these individuals at follow-up clinics is to evaluate their medical and functional status. Examination of medical status is important to review the progress of clinical status after discharge, identify red flags against an expected outcome, and early detection of any complications. Functional status evaluation helps to understand the recovery of functions lost during acute illness, the extent of remaining disability, and patients' individual needs to fulfill their activities of daily living (ADLs). Our strategies at follow-up should be directed to reduce hospital readmission and morbidity to make combined decisions for further treatment.

In this chapter, we will focus on the approach for evaluation at follow-up after critical illness. We will cover concepts behind general principles, which are commonly followed during consultations, including follow-up related history taking and explanation to patients.

CASE SCENARIO

Mrs XY, a 30-year-old bank employee, married for the last 6 months, sustained an alleged road traffic accident. She lost consciousness at the site of the accident, but was immediately brought to the emergency. On arrival at the emergency, she was opening her eyes only after shaking her, was unable to talk, and was moaning in pain. Imaging of the brain reported bleeding on the left side and she was undertaken for surgery (left decompressive craniectomy). Postoperatively, she was admitted to the ICU and was ventilated for a few days. She received feeds through a nasal tube and was on a urinary catheter. Later, she needed a tracheostomy and was shifted to a ward for 2–3 days. At discharge, she was able to move her left side. She was unable to speak because of the tube in her throat. Now, she has come for review at the follow-up clinic.

This is the history given by her husband who is accompanying her. How will you proceed with this consultation?

HISTORY TAKING

In general, history taking is directed toward understanding the course of events of symptoms to establish a diagnosis. History taking in follow-up clinics needs to be directed to find the consequences of illness and the treatment received during previous hospital admission. This can include details of the development of complications and morbidity after discharge, which should lead a clinician to plan further required evaluation. Ordinarily, patient history is obtained with an interview of the patient but in certain cases like in patients with cognitive or communication deficits, the clinician must try to obtain a reliable history of events after discharge from a close relative or a caretaker. There are two most important things to do after the initial history is given by the patient or caretaker; firstly, to find clinically relevant gaps in the history given by asking correct questions that will help us to understand the details of the severity of the illness. Secondly, the course of events that occurred after discharge to know if the patient has experienced any expected or unexpected complications and/or improvement, if any. The adverse thing a clinician can do is to assume answers to these gaps from their own previous experiences with patients. It is always helpful to confirm and reconfirm by asking to avoid bringing personal bias in evaluating patients.

Consider covering all aspects of history including chief presenting complaints, history of new complications or symptoms, past and personal history, presence of comorbidities, vocational details, and social and family history. Sleep patterns, addictions, diet preferences, etc., are known to change after a critical illness. Finally, functional history to estimate the effect of illness on ADLs is a crucial part of follow-up consultations.

Functional Assessment

Impairments in functions after a long critical illness are common. These mostly occur due to muscle-related weakness, impaired cognition, partial neurological recovery, or the chronic progressive nature of the illness. The ultimate aim of health care is to ensure a reduced burden of disease and community reintegration of patients. Functional assessment helps in putting impairments, activity limitations, and participation restrictions in an objective perspective for a quantifiable problem-based approach to the treatment of residual disabilities. There are multiple disease-specific or general tools available to assess functions that are validated for use in clinical as well as disability assessment. Even when one is not aware of these assessment tools, mobility status and involvement by the patient in ADLs can be easily assessed.

Mobility status can be noted as bedbound—bed mobility independent or dependent, wheelchair—self or caretaker driven, and walking—with or without support. It is important to notice walking distance—indoors or outdoors, use of stairs, safety, and risk of falls in patients who are mobile independently to prevent complications from falls.

Performing ADLs such as eating, drinking, dressing, using of bathroom, toilet, and bathing can be assessed while interviewing a patient or caregiver. Advanced ADLs such as cleaning, cooking, driving, performing hobbies, or sexual intercourse can be assessed when appropriate. All ADL functions can be classified easily as independent/partially dependent/completely dependent.

Before proceeding to the examination, reviewing the discharge summary will shed more light on clinical information such as discharge medications and guide a clinician through the flow and focus of the examination.

In the given case scenario, the following history of the acute presentation is missing:

- History of immediate, early, or late seizures
- History of any peripheral injuries
- Motor assessment at the time of presentation to the emergency department
- Need for long-term tracheostomy tube (TT)
- Medicolegal aspects of the case, if any

We will also need details of events after she was discharged from the hospital. Here, we can ask following details to understand the progression:

- Medications
- Change in her cognitive and mental status after discharge
- Appetite (veg/nonveg/mixed), number and frequency of feeds
- *Fluid intake/output:* Liter/day
- *Sleep:* Normal/reduced/increased
- *Tube care:* TT, urinary catheter, nasogastric (NG) tube
- History of fever/cough
- Status of her right-sided weakness
- *Education:* Literate/illiterate
- *Vocation:* Employed/unemployed/type of job
- *Addiction/habituation:* Yes/no
- *Menstrual cycle:* Regular/cycle duration/duration of bleeding
- *Contraception:* +/-
- Sexuality/fertility
- *Bladder:* Self/diapers/indwelling urethral catheter/indwelling suprapubic catheter (SPC)/urocondom
- *Bowel:* Regular/irregular (self/diapers/need for suppository/enema/laxatives)
- *Functional history:* This includes the following—
  - *Mobility:* Bed bound/ambulant (self/wheelchair)
  - *ADL:* Independent/dependent
- *Architectural history:* Barriers to accessibility at home/workplace +/-

- *Felt needs or expectations (patient/caregiver):* This is the most important aspect from the functional point of view and helps the physicians to work along with the patient and their families towards realistic goal setting.

The felt needs in her case could be one or all of the following:

- Walk independently
- Removal of all tubes (tracheostomy, NG, and indwelling urethral catheter)
- To rejoin work in the bank (as she is a bank employee)
- To resume her sexual life

Based on the history of Mrs XY, the clinician can proceed for examination with focus on neurological examination, along with evaluation of functional status.

EXAMINATION

The focus of the required examination should be clear from the history taken. The examination needs to be completed in an appropriate environment without causing significant distress to the patient, considering the privacy of the patient and the chaperon requirement as fit. It should aim at evaluating the general condition of the patient, screening for other conditions (not mentioned by the patient during history but expected after the illness), impairments, and disability. Outcome after critical illness is variable and depends on multiple factors such as severity of illness, premorbid condition of the patient, the timing for receiving treatment, and also the illness itself. This means that during follow-up, one may come across a broad spectrum of clinical presentations. Patients can present with minimal recovery, with secondary complications, or near complete to complete recovery.

Examination of a patient essentially consists of general examination, systemic examination followed by focused examination, and special tests related to primary illness.

General Examination

The general examination is a combination of checking vitals, observation/inspection of important external signs of internal pathology, general appearance, and examination of skin, lymphatics, and vascular system **(Table 1)**. A clinician can consider checking for blood sugar level, lung capacity with a flow meter, etc., depending on the presentation in the outpatient department (OPD; **Table 2**). The examination will also include noting various tubes—in situ and related inspections when applicable **(Table 3)**.

How to assess tubes?

The NG tube, TT, percutaneous endoscopic gastrostomy (PEG), SPC, per urethral indwelling catheter (PU catheter), and urocondom are the most observed medical tubes during OPD follow-up. At times, patients may be on home ventilators, noninvasive ventilators (BiPAP and CPAP), or portable oxygen through various ports. It is essential to examine tubes present in situ to check functioning, hygiene or cleanliness, any related complications or even patients'/caregivers' understanding of the care of these tubes **(Table 3)**.

In the given case scenario, the examination should include the following:

- Vitals (resolution or persistence of autonomic dysfunction)
- Oxygen saturation
- Pain (musculoskeletal/neuropathic)
- *Skin:* Surgical site/wounds (pressure ulcer)
- Weight
- Tubes in-situ (TT/NG/PU catheter)
- Orientation (person/place/time)

Systemic Examination

Next part of the examination is systematic review of all the systems with the help of inspection, palpation, auscultation, percussion, and special tests when indicated **(Table 2)**. The systemic examination during critical presentation is covered in Section 2 of this book. Although the primary pathology does not change during follow-up consultation and the system that needs to be examined in detail remains the same, there are certain unique aspects of clinical presentations at follow-up that need to be kept in mind. These unique presentations are the result of medical management during critical illness and the effect of natural progression of the disease. **Table 2** summarizes such unique examinations to follow for each system. These examinations are to be performed over and above routine examinations for a particular system.

In the given case scenario, the systemic examination should include the following:

- *Higher mental functions:* Conscious, visual fixation/tracking, obeying simple one-step or two-step commands (consistent/inconsistent), identifying letters/numbers/common objects, and using a mobile phone with the left hand.

TABLE 1: General examination.

General examination	***Look for***	***Special consideration***
General appearance	Sick/uncomfortable/healthy/distressed/disconnected/abnormal postures	• Abnormal postures can present with brain injury • Decorticate (flexor posturing) • Decerebrate (extensor posturing)
Built	Weight/body mass index	Patients with impaired cognition/dysphagia may not receive adequate nutrition
Level of consciousness	Awake/alert/orientation/mood	For neurological illness, detailed evaluation can be performed later
Saturation	Room air/on oxygen	• Noninvasive ventilation (BiPAP and CPAP) • Tracheostomy tube (type/size of tube/secretions)
Respiratory rate	Observe for a minute or use a pulse oximeter	Note the position in which rate is observed
Temperature		Autonomic dysfunction
Pulse rate	Notice rate, rhythm, volume, and equality on both arms	Autonomic dysfunction
Blood pressure		Autonomic dysfunction, postural hypotension
Pain	Estimate pain intensity with scales (wherever possible)	Differentiate between various types of pain (musculoskeletal/neuropathic)
Pallor/icterus	Principles as per routine general examination	
Nails, tongue		
Lymphadenopathy		
Skin condition	Surgical wounds, pressure ulcers, and diaper rash	Site and stage of pressure ulcer
Lymphatic system	Lymphedema	Pitting or nonpitting and notice extent of edema
Vascular system	Peripheral pulses, deep vein thrombosis	
Tubes-in-situ	Note condition and related issues	
Limbs	Deformities, contractures, and loss of part or the whole limb	

(BiPAP: bilevel positive airway pressure; CPAP: continuous positive airway pressure)

TABLE 2: Systemic examination.

Central nervous system	
Pupils	Pupils are the doorway to the brain. The examination helps in evaluating current clinical status and prognostication
CNs	• Clubbing the nerve is the most effective way for examining of CN I, II, (III, IV, and VI) together, V, VII, VIII, (IX and X) together, XI, XII • Note down residual deficits
Language and speech	• Naming/repetition/comprehension/fluency/paraphasias/prosody/articulation/reading/writing • Remember that neurogenic communication disorders such as aphasia are different from dysarthria or apraxia of speech • Dysarthria is pure neurogenic in origin, which is different from speech difficulty due to structural etiology • Aphasia is a language disorder affecting reading, writing, auditory comprehension, or verbal output • Remember to look and recognize the deficits. Detailed examination is out of the purview of this book
Higher mental functions	• *Level of consciousness:* GCS—EVM/alertness • *Orientation:* Time/place/person • *Memory:* Immediate/recent/remote • Language and speech • *Apraxia/neglect/attention/calculation:* +/- • There are various scales for the assessment of cognitive functions such as the MMSE, MoCA, and ACE-R

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Central nervous system	
Swallow	• Dysphagia is common after most neurological conditions. New onset of dysphagia in follow-up visits can be present in progressive neurological conditions. Patients who were discharged with NG tube in situ will need swallow evaluation at the follow-up consultation. Checking for signs of aspiration with auscultation and radiographs, and when indicated, is an important aspect of evaluating swallow, more so when patients try eating without medical clearance or guidance at home • Remember to look and recognize the deficits
Motor examination	• Observing any muscular atrophy, change in tone and/or ROM of all joints, muscle strength remains mainstay • Remember that motor examination at follow-up needs to focus on exploring for deformities or contractures (reduced ROM) and changes in tone (spasticity, rigidity, and hypotonia). Change in muscle tone is expected as a part of natural recovery and no change from hypotonia is usually considered as a sign of poor prognosis • The most common deformities of the upper limb after a neurological injury are flexion of the upper limb (elbow, wrist, and fingers). In the lower limb, the flexion deformity of the hip, knee, and ankle (equinus deformity) can be seen • The deformities can be static (at rest) or dynamic (while performing a motor task such as standing/walking). The range of motion needs to be performed across all major joints (shoulder, elbow, wrist, hip, knee, and ankle) to conclude if these deformities are fixed (called as contractures) or correctable • An extremity ultrasound might be required (to supplement the clinical examination) to confirm the diagnosis related to musculoskeletal conditions such as muscle tear, joint effusion, tendonitis, etc.
Sensory examination	• Principles and steps for sensory examination are the same. Areas affected and type of sensory deficits need to be explicitly explained to patients to avoid complications. Loss of touch, temperature can cause burns, loss of joint position sense causes balance issues and so increased risk of fall, which directly can affect mobility of the patient • Remember to discuss implications of all types of sensory deficits with patients to avoid related secondary complications
Bowel/bladder issues	• Involvement of bladder and bowel function after neurogenic illness is known. The most easy way to evaluate involvement of bowel and bladder is thorough history exploring urge and control of both functions • A renal ultrasound with prevoid and postvoid is an important investigation in evaluating all neurogenic bladders once the patient starts self-voiding • Remember to perform relevant examinations. For example, due to bowel involvement after spinal cord injury, patients may develop hemorrhoids or fissures due to long-term constipation. Per-rectal examination can prognosticate and indicate recovery, etc.
Cardiovascular and respiratory system	
Even with the primary involvement of either of the cardiovascular or respiratory systems, other systems must be examined with equal attention because of close physiological association between these two systems. There are multiple peripheral signs pointing to the involvement of the cardiovascular or respiratory system. The extent of these involvement can be an indirect indicator of recovery and must be explored during general examination. Following are a few of the special tests that can be performed in follow-up OPD along with routine inspection, palpation, auscultation, and percussion	
Single breath count	This is performed by asking patients to take a deep breath and then count as much as possible in their normal speaking voice without taking another breath. Single breath count <15 usually indicates low FVC and respiratory muscle weakness
Peak flow meter	This can be used primarily after respiratory conditions as an indicator for lung capacity
Ankle brachial index	In primary or secondary peripheral vascular diseases, it is a good indicator of blood circulation and it serves as prognostic marker for cardiovascular events even in absence of symptoms
Point-of-care investigations depending on the availability of resources are helpful in review of clinical status at follow-up	
Orthopedic examination	
This examination should focus on looking for contractures, impairments, and functional deficits **(Figs. 1 and 2)**. Motor and sensory examination can be conducted on these lines similar to explained in central nervous system examination	

(ACE-R: Addenbrooke's Cognitive Examination-Revised; CN: cranial nerve; EVM: eye, verbal, and motor; FVC: forced vital capacity; GCS: Glasgow Coma Scale; MMSE: mini-mental state examination; MoCA: Montreal Cognitive Assessment; NG: nasogastric; OPD: outpatient department; ROM: range of motion)

TABLE 3: Examination of tubes.

Tube in situ	***Look for***
Nasogastric tube Percutaneous endoscopic gastrostomy	• Tube size, stains, and hygiene of the tube, positioning • Pressure necrosis or ulceration of surrounding tissue • Review feeds given, rough calorie intake
Tracheostomy	Tube type (cuffed/uncuffed), size, inner tube, secretions, frequency of suctioning, bleeding (over granulation)
Urinary catheter	• Tube type and size, anchoring over lower abdomen in males, to thigh or lower abdomen in females • Hygiene and urinary bag examination • Pressure necrosis or ulceration of surrounding tissue (penoscrotal fistula)

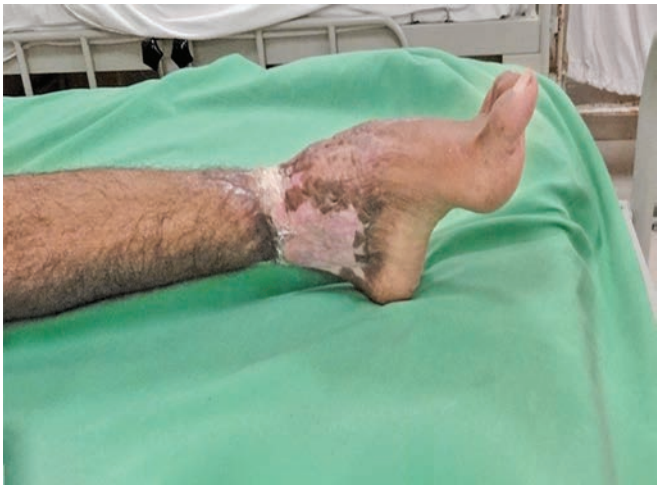

Fig. 1: Fixed ankle deformity, loss of muscles after electrocution injury.

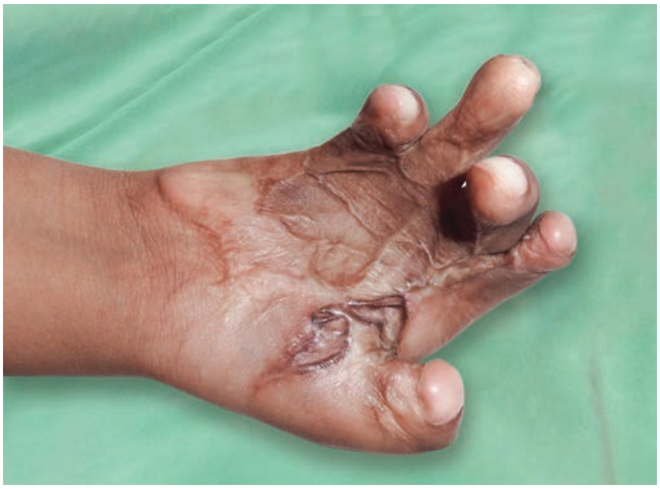

Fig. 2: Postburn contracture of right palmar fascia.

- Motor examination of the right upper and lower limb, which would include the following:
  - *Tone:* Increased (most likely), normal or reduced tone
  - Range of motion across major joints (restricted/full/pain present/absent)
  - *Deformities:* Flexion contracture of upper and lower limb (Distal >proximal)
  - *Power (Examine flexion/extension in major muscle groups):* >3/5 or <3/5
  - *Reflexes:* Mostly brisk/exaggerated
- Swallow assessment (if cognition is good) by a swallow therapist
- Tubes in situ
- Bladder/bowel

SUMMARY

It is a good practice to explore patient's (and family's) ideas about the illness and concerns such as improvement, finances, and expectations from consultation and treatment at the end of the examination. This discussion is more effective if placed after examination as the clinician is in a better position to answer questions regarding the current clinical picture, prognosis or needed investigations, treatment, and its feasibility. This, in turn, helps in combined decision making and acceptable management plan. One should not shy away from referring patients to other specialist colleagues for their input. It is always important to make use of expertise and facilities available at the hospital for the benefit of patients. The chances of disappointments or arguments from either side significantly reduce with healthy, open conversations. Learning soft skills, knowing red flags, conveying difficult medical language in simpler understandable words, asking for help at the right time from seniors or other clinicians are all integral parts of follow-up consultation. For a successful consultation at follow-up, one must understand the key differences in examination goals during critical illness and at follow-up, which not only focuses on the disease part but also equally on identifying quality of life, impairment, and disabilities.

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